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Headings and sub-heads should not be underlined. Binomial specific names and other words to be printed in italics should have a dotted underline.

Tables should be limited and be typed on separate sheets of paper numbered consecutively, Table 1, Table 2, etc. Figures, including photographic prints, graphs, maps, etc. should be numbered consecutively, Fig. 1, Fig. 2, etc., and attached at the end of the text. References to tables and figures in the text should be by number and not to "table below" or "figure below". Coloured illustrations are reproduced only at the author's expense.

Bibliographical references should be listed in alphabetical order of first author or country (if annual report) at the end of the paper, and not numbered. Only those cited in the text should be included. References cited in the text should be inserted, as e.g. (Richards 1950) or "Richards (1950) showed".

If the same author is cited more than once, his publications should be arranged in chronological order in the list of references, and if more than one publication of the same author in the same year of publication is included, the letters "a, b, c" should be added after the date in both the list of references and in the text.

References should include, in the following order, surname, initials of author(s), year of publication (in parentheses), World List abbreviation of title of periodical (dotted underline), volume number (arabic numerals underlined), first page number. The title of the article should not be included.

References to books should include name and initials of author(s), year of publication (in parentheses), the exact title (underlined), town of publication, page number (if page number specifically cited).

References to annual reports should state the country, year of reference, followed by the name of the department or organisation, e.g. Kenya (1955) An. Rep. Dept. Vet. Services, p. 50 (if specific page cited).

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STUDIES OF PARASITIC HELMINTHS OF SHEEP AND GOATS IN GHANA

R.K.G. ASSOKU,
Department of Animal Science, University of Ghana, Legon, Accra, Ghana.

SUMMARY

The incidence, occurrence and identity of helminth parasites in sheep and goats under diverse management and husbandry practices on the Accra-Plains of Ghana, were studied over a period of 8 months.

Nine hundred and eight sheep and 360 goats, obtained from farms, villages and towns scattered within, and covering an area of about 500 square kilometres of the plains were examined coprologically.

The results generally showed a relatively high incidence of helminth load in both the sheep (80%) and goats (88.3%) in the country, particularly during the first year of life. A total of 12 nematodes and one each of cestode and trematode, and 10 nematodes and the same species of cestode and trematode, were isolated and identified in sheep and goats, respectively. The commonest ovine and caprine helminths identified were, respectively, Ostertagia circumcinta and Trichostrongylus axei, and although most of the helminths identified in this study have already been reported from Ghana and/or West Africa, Cooperia curticei, Gaigeria pachyscelis, Nematodirus filicollis and Ostertagia mashaiki are being reported for the first time in sheep and goats in Ghana, the latter two nematodes in sheep only.

The overall results of these studies, which also examined the effects and influence of the age, sex, management practices, nutrition, and breed of the animals on the incidence of, and susceptibility to, helminth load in these important food animals, were also discussed in relation to the improvement, management and the viability of this rapidly expanding industry in Ghana.

INTRODUCTION

Sheep and goats play an important role in Ghana in providing animal protein in the diet, and together they also provide a large proportion of the meat that is consumed. During recent years, there have been tremendous advances in the small ruminant industry in various parts of the country, and the more extensive native methods of management, particularly for goats, are giving way to the more intensive modern methods of production. Unfortunately, some of these modern methods of management expose these animals to environmental and other conditions involving high risk of parasitism.

No study had previously been undertaken in this country to ascertain the importance of helminth infections in these food animals, or to record the parasite distribution, except a list of parasites and losses caused by helminths in sheep and goats which were briefly reported by Fry (1958), Edwards and Wilson (1958), Jackson (1965) and Oppong (1973). The present studies were therefore undertaken to fill this gap, especially to obtain a general profile of the helminth species occurring in sheep and goats on the Accra-Plains of Ghana — perhaps one of the most important livestock producing areas in Ghana (Assoku, 1979). The present studies also involved estimating both
the general incidence and examining the influence of those factors that affect the rise, distribution, occurrence and susceptibility of ovine and caprine helminth parasites in Ghana.

**MATERIALS AND METHODS**

**Location:** The studies were concentrated on the north-eastern part of the Accra Plains of Ghana. The area, geography and weather of this region have already been described by Assoku (1979). The studies covered the period from June to January, and this included part of the mini rainy season. Flocks of sheep on three livestock research farms and in six villages and towns scattered within these plains were examined. The various centres were chosen because of their accessibility and the different types of management procedures being practised there.

**Animals:** Animals sampled were normally 2 months of age and above. Three distinct types (or breeds) of sheep and two of goats were studied. These consisted of West African (W.A.) Dwarf or the local Forest type sheep, W.A. Long-Legged Sheep and the Nungua Black-Head sheep (N.B.H.); the W.A. Dwarf and the W.A. Long-Legged goats were also examined. Except for the N.B.H. sheep which is a new breed evolved locally from the Black-Headed Persian and the Forest type (Ngere, 1973) the rest of the sheep and goats constitute the main indigenous types or breeds of small ruminants on the Accra-Plains. Since the investigation was also designed to study the incidence and pattern of distribution of the natural infection under the different farming conditions and practices, great care was taken not to disturb or interfere with normal flock management. Particulars of each animals, such as age, sex, diet, breed (type), health status, lactation characteristics, time of last lambing/kidding and the state or method of management, were all recorded.

**Faecal examination:** Faecal samples were taken directly from the rectum of each animal by manual manipulation, using disposable rubber gloves. The samples were then transferred to the laboratory in special numbered containers, and worm-egg counts were performed using the McMaster technique as outlined by Gordon and Whitlock (1939).

Identification of the various helminth parasites was made from both the morphology of the egg and with the aid of a calibrated eyepiece-micrometre and the helminthological chart prepared by Cunliff and Crofton (1954). Those faecal samples from which definitive identification of the helminths could not be made from the morphology of the egg and others were subjected to coproculture and eventual identification of the resulting larvae harvested from such eggs by the Baerman's technique (Wetzel, 1930) after prior incubation at 25°C for 7-14 days. The helminthological chart prepared by Dickmans and Andrews (1933) was used for the larval identification.

**Analysis of data:** The "t" statistics as explained by Alder (1968) and Sokal and Rohlf (1936) were used to determine differences between observed values with reference to the age, breed, sex, nutritional status, lactation (post-parturient) characteristics, the management and husbandry practices in those areas where the animals were found and the parasites identified.

**RESULTS**

A total of 908 sheep and 360 goats were coprologically examined during this
study. In general, the results showed a relatively high worm load in both sheep and goats in the country; 726 (80.0%) and 381 (88.3%), of the total numbers of sheep and goats respectively. However, single helminths infection was detected in only 12.12% and 13.2% of the total infected sheep and goats, respectively, indicating a high mixed-infection rates in both species.

Types of helminths isolated and identified: The survey established the presence of a total of 12 nematodes and one each of cestode and trematode in sheep. In goats, 10 nematodes and the same species of cestode and trematode were isolated and identified. All these parasites, isolated and identified in sheep and goats and listed in decreasing order of incidence, are summarised in Table 1. The commonest ovine and caprine helminths, respectively, were Ostertagia circumcincta (30.9%) and Trichostrongylus axei (28.8%). The cestode, Moniezia expansa, was ubiquitous in both the sheep and the goat, occurring singly, usually in young animals, and only occasionally in mixed helminth infections.

Cooperia curticei, Gaigeria pachyscelis, Ostertagia mshali and Nematodirus filicolis are being reported for the first time in this country, the latter two in sheep only.

Table 1: Incidence and types of helminths isolated in sheep and goats on Accra-Plains of Ghana

<table>
<thead>
<tr>
<th>Sheeps</th>
<th>%</th>
<th>Goats</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ostertagia circumcinta</td>
<td>30.9</td>
<td>Trichostrongylus axei</td>
<td>28.7</td>
</tr>
<tr>
<td>Trichostrongylus axei</td>
<td>26.6</td>
<td>Ostertagia circumcinta</td>
<td>27.3</td>
</tr>
<tr>
<td>Haemonchus contortus</td>
<td>12.5</td>
<td>Haemonchus contortus</td>
<td>21.0</td>
</tr>
<tr>
<td>Trichostrongylus colubriformis</td>
<td>7.4</td>
<td>Trichostrongylus colubriformis</td>
<td>5.5</td>
</tr>
<tr>
<td>Cooperia curticei</td>
<td>5.4</td>
<td>Moniezia expansa*</td>
<td>4.6</td>
</tr>
<tr>
<td>Chabertia ovina</td>
<td>4.9</td>
<td>Chabertia ovina</td>
<td>4.5</td>
</tr>
<tr>
<td>Strongyloides papillosus</td>
<td>2.8</td>
<td>Strongyloides papillosus</td>
<td>3.2</td>
</tr>
<tr>
<td>*Moniezia expansa</td>
<td>2.0</td>
<td>Cooperia curticei</td>
<td>1.6</td>
</tr>
<tr>
<td>Gaigeria pachyscelis</td>
<td>1.3</td>
<td>Gaigeria pachyscelis</td>
<td>1.1</td>
</tr>
<tr>
<td>+Fasciola gigantica</td>
<td>0.9</td>
<td>Oesophagostomum columbianum</td>
<td>1.0</td>
</tr>
<tr>
<td>Oesophagostomum columbianum</td>
<td>0.8</td>
<td>Fasciola gigantica+</td>
<td>0.5</td>
</tr>
<tr>
<td>Ostertagia mshali</td>
<td>0.1</td>
<td>Trichuris ovis</td>
<td>0.3</td>
</tr>
<tr>
<td>Nematodirus filicolis</td>
<td>0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trichuris ovis</td>
<td>0.03</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* — Cestode
+ — Trematode
The relationship between age and helminth load: The variation and distribution of faecal helminth load with age in the different types or breeds of sheep and goats are illustrated in Fig. 1. There was a steady decline in average worm-egg output during the first year of life, after which age the worm load stabilised, the level of infection remaining steady and relatively low. The indigenous sheep (W.A. Dwarf or local Forest) and goat (W.A. Dwarf) however were more liable to become very heavily infected with parasites early in life, but as they grew older, they gradually and significantly showed decreased helminth loads. Indeed, these indigenous breeds became completely helminth-free by the time they were about 4 years old (Fig. 1).

An idea of the relative resistance of the indigenous breeds to helminthiasis could also be discerned by the finding that some of these animals harboured worm loads as high as between 38,000 and 63,000 eggs per gram (EPG) and yet easily survived clinical disease, whereas normally worm loads of over 23,000 E.P.G. invariably cause clinical disease and death (Edwards and Wilson, 1958). In contrast, the susceptibility of the only non-indigenous breed of sheep studied (i.e. NBH) was clearly demonstrated by the persistence of a relatively high faecal helminth load over the years which actually tended to increase after four years (Fig. 1). These observations clearly illustrate the adaptability and breed susceptibility of these animals to helminth infection.

---

Fig. 1: Distribution faecal helminth load with age in the different breeds of sheep and goats

- **W.A. DWARF (FOREST) SHEEP**
- **W.A. LONG-LEGGED SHEEP**
- **NUNGA BLACK-HEAD (N.B.H) SHEEP**
- **W.A. DWARF GOAT**
- **W.A. LONG-LEGGED GOAT**

**Mean Egg Count (E.P.G.)**

<table>
<thead>
<tr>
<th>Age (in months)</th>
<th>1-6</th>
<th>7-12</th>
<th>13-18</th>
<th>19-24</th>
<th>25-30</th>
<th>31-36</th>
<th>37-42</th>
<th>43-48</th>
<th>49-54</th>
<th>55-60</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1000</td>
<td>2000</td>
<td>3000</td>
<td>4000</td>
<td>5000</td>
<td>6000</td>
<td>7000</td>
<td>8000</td>
<td>9000</td>
<td>10000</td>
</tr>
</tbody>
</table>
The effect of supplemental feeding on mean worm-load in sheep: Table 2 shows the effect of supplemental feeding on the mean worm-egg load in sheep. Some of the animals on natural pasture were supplemented with green feed and concentrates, the former consisting mainly of cassava peels and Giant-star grass; the control group had no supplementation. In both age groups (i.e. lambs and adults), the supplemented group of sheep carried significantly heavier (P<0.01) helminth infestation than the control, unsupplemented group (Table 2).

Table 2: The Effect of supplemental feeding on mean-worm (Helminth) load in sheep.

<table>
<thead>
<tr>
<th>Animals</th>
<th>Mean Worm-Egg Load (EPG)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supplemented Group</td>
</tr>
<tr>
<td>Lambs</td>
<td>$6426^+$ (148)</td>
</tr>
<tr>
<td>Adults</td>
<td>$641^{++}$ (57)</td>
</tr>
</tbody>
</table>

$^+$ — Differences are significant at 1% (P < 0.01)

Effects of pregnancy and lactation on helminth load in sheep and goats:

The characteristic increase in faecal worm-egg load as a classic example of the effect of stress factors like lactation (i.e. the so-called post-parturient or lactational egg rise) on helminth load (Crofton, 1958; Dunsmore, 1965; Connan, 1968) was exemplified by the significantly higher (P<0.01) faecal egg counts in lactating female sheep and goats than in their non-lactating and pregnant counterparts (Table 4). There

Table 3: The Effect of the type of management/husbandry practices on the mean worm-egg load in sheep

<table>
<thead>
<tr>
<th>Animals</th>
<th>Mean Worm-Egg Load (EPG)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Extensive System (free range grazing) with irregular drenching schedules</td>
</tr>
<tr>
<td>Lambs</td>
<td>9813* (96)</td>
</tr>
<tr>
<td>Adults</td>
<td>763** (102)</td>
</tr>
</tbody>
</table>

* ) — Differences are significant
** ) at 1% (P<0.01)

Figures in brackets are the total number of animals examined in each group.

Effect of management/husbandry practices on worm-load in sheep: The studies also examined the effect of management and other husbandry practices on the degree of worm infestation. Table 3 shows the mean worm-egg counts for both lambs and adult sheep which indicates that, as expected, sheep kept under rotational or restricted grazing (semi-intensive) system of management had fewer worm-egg counts (P<0.01) than those kept under the free-range grazing or extensive system.
was no significance \((P > 0.05)\) however in the faecal egg counts between non-lactating and pregnant ewes and does, between non-lactating ewes and does and between pregnant ewes and does. It was also established that the commonest helminth species contributing to the high faecal egg counts in lactating animals were *Ostertagia circumcinta*, *Haemonchus contortus* and *Trichostrongylus* spp.

Table 4: Effects of Pregnancy and Lactation on the Total Worm-Load in Ewes and Does

<table>
<thead>
<tr>
<th>Animals</th>
<th>Lactating Animals</th>
<th>Mean Worm Non-Lactating Animals</th>
<th>Egg-Load Pregnant Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheep</td>
<td>6112* (65)</td>
<td>1197* (49)</td>
<td>842* (54)</td>
</tr>
<tr>
<td>Goats</td>
<td>4900** (46)</td>
<td>1015** (16)</td>
<td>1396** (38)</td>
</tr>
</tbody>
</table>

* ) — Differences are significant ** ) at 1% \((P < 0.01)\)

Figures in brackets are the total number of animals examined in each group

The effect of the duration of lactation on the worm-egg load in lactating ewes is also illustrated in Fig. 2. Increase in lactation period resulted in decreased helminth load, the high infestation normally seen in lactating animals being present only during the first two weeks of lactation.

The effect of sex of animal on mean worm-load: Table 5 summarises the data on the analysis of the total worm-egg counts obtained for the various sexes and age groups of sheep and goats. The results showed that ram lambs had significantly greater \((P < 0.01)\) total worm-egg counts than ewe lambs. There was, however, no significant difference \((P > 0.05)\) between the counts in both sexes of adult sheep. The results also showed that whereas buck kids carried significantly lighter \((P < 0.01)\) helminth load than the doe kids, adult bucks had much greater \((P < 0.01)\) mean worm-egg load than does.
Table 5: The Effect of Sex on the Mean Worm-egg counts of sheep and goats

<table>
<thead>
<tr>
<th></th>
<th>Sheep</th>
<th></th>
<th>Goats</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>Mean Worm-Egg Load (EPG)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lambs/Kids</td>
<td>8187*</td>
<td>4088*</td>
<td>1608+</td>
<td>3487+</td>
</tr>
<tr>
<td></td>
<td>(80)</td>
<td>(84)</td>
<td>(13)</td>
<td>(2)</td>
</tr>
<tr>
<td>Adult</td>
<td>994**</td>
<td>1013**</td>
<td>4139*</td>
<td>1327*</td>
</tr>
<tr>
<td></td>
<td>(63)</td>
<td>(52)</td>
<td>(42)</td>
<td>(55)</td>
</tr>
</tbody>
</table>

*, +, *, — Difference was significant at 1% (P < 0.01)
** Difference was not significant at 5% (P > 0.05)

Figures in brackets indicate the number of animals examined.

DISCUSSION

The present study has shown that a reasonably high percentage of the small ruminants found on the Accra-Plains of Ghana were infected with helminth parasites, but the degree of infection and/or worm-load greatly depended on the levels of management and hygiene obtaining in those areas.

Most of the parasites isolated in this study have previously been isolated and reported from Ghana and/or West Africa (Beal, 1929; Edwards & Wilson, 1958; Jackson, 1965; Doku, 1972; Naate, 1973), but Cooperia curticei, Gaigeria pachyscelis, Nematodirus filicoli and Ostertagia mashali are being reported for the first time in Ghana, the latter two in sheep only.

Jackson (1965) had enumerated the types of parasites that were likely to occur in domestic animals in West Africa but had made no definite mention of any of those helminths known to be present in Ghana. However, the studies of Doku (1972) and Naate (1973) had indicated that Haemonchus contortus, Ostertagia circumcincta, Trichuris ovis, Trichostrongylus spp., Chabertia ovina, Moniezia expansa and Strongyloides papillosus were present in sheep and goats in certain areas of the Accra-Plains.

As has previously been reported by Fabiyi (1970) from Nigeria and Jackson (1975) from Ghana, the present studies also indicate that Moniezia expansa can be found mostly in young animals, and the time of minimum occurrence of this cestode in a host coincided with the period of heaviest worm-load of other helminths, and vice-versa. This observation may be the result of one or more of the other helminths having a suppressive or inhibiting effect on M. expansa (Fabiyi, 1970).

The commonest ovine and caprine helminths isolated and identified in this study were, respectively, O. circumcincta and T. axei. This finding is in contrast with the previous observations of Beal (1929) and Oppong (1973), who reported that H. contortus was the most predominant helminth parasite found in both sheep and goats in Ghana. This discrepancy could be explained by the observation that since H. contortus is readily killed by many of the common anthelmintics (Gordon, 1950) the routine and regular use of such drugs on some of the farms studied might have eliminated most of this particular helminth. This explanation is supported by the finding that in two of the areas studied, where anthelmintics were sparingly, if ever, used, the commonest helminth isolated in both areas was indeed H. contortus (33.8% and 41.7%, respectively).
The observation that there was a decrease in worm-load or total egg count in both the sheep and goats with age is not an uncommon finding in such studies (Goldberge, 1952; Paver, et al. 1955; Edwards & Wilson, 1958; Brunsdon, 1962), and Gordon (1950) has considered the increased resistance to infection and/or re-infection with age to be due to immunity as a result of the intake of small numbers of larvae early in life. It is probable therefore that this naturally acquired immunity, unlike the phenomenon of "self-cure" (Stewart, 1953) may be more important in the resistance of these animals to helminthiasis. Other non-immunological factors have however been described as being responsible for this increased resistance to helminth infection with age (Herlich, 1960).

The finding also that the indigenous breeds of sheep had a relatively lower worm-load than the exotic-local crosses was consistent with the earlier observation of Edwards and Wilson (1958) that the progeny of such crosses (i.e. the N.B.H. sheep) had always been shown to be more susceptible to heavy worm infestation than the local, indigenous breeds of sheep. These differences in the worm-egg counts in the different breeds of sheep and goats might thus be considered a measure of the resistance or susceptibility of the various breeds to helminthiasis, and the differences in resistance or susceptibility may probably be due to the indigenous breeds being more fully acclimatized to their peculiar environment (Stewart, Miller & Douglas, 1937; Golglazier et al. 1968) than the local-exotic crosses. On the other hand, the finding of a greater degree of infestation in the young local indigenous animal, as opposed to the adult, might really be due to a greater and/or earlier grazing activity by the young because their dams normally have poor milk supply (Spedding et al. 1964).

It has long been known that sheep and goats, on a consistently high plane of nutrition, normally harbour fewer intestinal parasites than those on a comparatively lower plane of nutrition (Clunies-Ross & Gordon, 1933; Taylor, 1943; Whitlock, 1949). However, the results of this study are in sharp conflict with these observations, since sheep receiving supplementation showed significantly (P<0.01) greater or higher worm load than those without supplementation. A plausible explanation of this finding is that there was abundant grass for all the animals at the time of this study, and it would appear that the animals consumed adequate amounts for maintenance and growth, rendering superfluous any supplemental feeding. The greater helminth infestation recorded in the supplemented group of animals could therefore be due to the methods or mode of presentation of the supplemental feed to the animals since the supplement was presented in either very low feeding troughs or on the floor, so that faecal contamination of the feed easily occurred. The supplemented group of animals therefore had an additional source of infection, apart from those helminth eggs picked at grazing.

The finding that those animals that were kept under rotational, rather than free-range, grazing system had fewer egg counts was not unexpected, because this was related to the methods of husbandry practices obtaining at the various farms studied, the single most important determining factor being the practice of drenching with anthelmintics. Quite clearly, the routine prophylactic,
drenching at regular intervals with different anthelmintics had a significant "lowering" effect on the total worm load in the respective animals on the various farms (Golglazier et al., 1968).

In this study, a "post-parturient rise" in faecal egg count occurred in all the lactating ewes and does, with Ostertagia spp. predominating. This finding is in agreement with those of Crofton (1954), Connan (1968) and Southcott et al. (1972) who reported of a dramatic rise in the total worm-egg count soon after parturition. An explanation for this phenomenon has been the subject of much discussion, but it is now generally agreed that it might be due to an increased fecundity in adult worms already in the alimentary tract (Parnell et al., 1954; Connan, 1968). It is also possible that as a result of the stress factors of lambing and lactation, the "post-parturient rise" may be the result of a generalized, non-specific, non-immunological loss of resistance by the ewe and doe to the nematode parasites in the alimentary tract. A similar explanation has, indeed, been variously offered by Parnell et al., (1954), Crofton (1958), Connan (1968) and Southcott et al. (1972).

The present studies have also shown that generally ram lambs harboured more helminth parasites than ewe lambs. This finding is similar to the previous observations of Golglazier et al. (1968), Vegors et al. (1971) and Knight et al. (1972), but it is in direct contrast with the findings of Scrivner (1964) who showed that ewe lambs in general harboured higher worm loads than ram lambs. Many theories have been offered to explain the host-sex effect on helminth parasites in sheep and goats. Interestingly, the finding that female hormones have an inhibiting effect on helminth parasites, in contrast with male hormones which have no such effect (Knight, et al. 1972), is most significant as it offers a plausible explanation for the significantly lower worm-egg counts found in ewe lambs. Nevertheless, the possibility that non-hormonal factors might also be operating in the present situation is confirmed by such conflicting observations as, unlike the sheep, buck kids carried significantly lighter helminth load than the doe kids (and vice-versa in the adult).

Many factors have been found to influence the cause, incidence and epidemiology of helminthiasis in sheep and goats. The present findings involving the age, sex, management practices, nutrition and breed of these important food animals are but a small contribution to the improvement, management and viability of a rapidly expanding industry in Ghana.

ACKNOWLEDGEMENTS

The author is very grateful to Mr. A.N. Akwoviah for his invaluable help in the collection of the data used in this study. The excellent technical assistance of Mr. S.K. Quarrantey is also gratefully acknowledged. The funds for this study were provided by the University of Ghana.

REFERENCES


Received for publication on 29th March, 1980
COMPARATIVE STUDIES OF THE INDIRECT HAEMAGGLUTINATION AND THE INDIRECT FLUORESCENT ANTIBODY TESTS IN THE DIAGNOSIS OF BOVINE CYSTICERCOSIS.

J.M. GATHUMA and P.G. WAIYAKI,
Faculty of Veterinary Medicine, P.O. Box 29053, Kabebe, Kenya.

SUMMARY

Results of the indirect haemagglutination (IHA) and the indirect fluorescent antibody (IFA) tests using sera from seven experimentally infected and four non-infected calves as well as 285 naturally infected and non-infected adult slaughter cattle were compared. Most calves showed detectable antibody responses 2—4 weeks post-infection.

The mean IFA titres in the experimental calves were slightly higher than the mean IHA titres using either crude antigen or fraction FI, the first fraction obtained by Sephadex G-200 exclusion chromatography of the crude antigen. Using sera from naturally infected and non-infected adult slaughter cattle, the IFA test was found to be more sensitive and more specific than the IHA test using crude antigen. However, there were no significant differences between the sensitivities and specificities of the two tests when FI antigen was used in the IHA test.

INTRODUCTION

The application of the indirect haemagglutination (IHA) and the indirect fluorescent antibody (IFA) tests in the diagnosis of bovine cysticercosis caused by the larval stage of *Taenia saginata* (*Cysticercus bovis*) has been tried by several workers (Walther and Goss-klaus, 1972; Calamel and Soul, 1972; Machnicka, 1973; Rydzewski *et al.*, 1975; Gathuma *et al.*, 1978). Results obtained by use of these two tests have so far been encouraging. Machnicka (1973) reported higher titres with the indirect immunofluorescence (IFA) test using activated *T. saginata* oncospheres as antigen than with the IHA test.

However, she used only a small number of experimental calf sera. Rydzewski *et al.* (1975) compared the IFA using paraffin sections of *T. solium* as antigen, the IHA and the agar gel precipitin tests in the immunodiagnosis of *T. solium* cysticercosis in humans. They found the IFA more specific than the IHA test. The gel precipitin test was the least sensitive. Cross-reactions with sera of individuals with *Echinococcus granulosus* infections (hydatid cysts) were observed in all the three tests.

The present study evaluates and compares the IHA and IFA tests in the diagnosis of bovine cysticercosis in experimental and natural infections.

MATERIALS AND METHODS

Animals

Three uninfected bovine calves (numbers 51, 60 and 70) and calf number 68 which was orally dosed with 150,000 *T. saginata* eggs but which, on postmortem examination, turned out not to have taken the infection, were used as negative controls. Calves 53, 54, 55, 61, 63, 65 and 7433 were infected. Details on their age, breed, infection regimen and necropsy findings are given in Table 1.
Table 1: Experimental calves: Breed, age, egg dose and post-mortem findings.

<table>
<thead>
<tr>
<th>Calf</th>
<th>Breed</th>
<th>Age at infection (months)</th>
<th>Egg dose (oral)</th>
<th>Duration of &quot;infection&quot; (days)</th>
<th>No. of cysts at P.M.</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>51</td>
<td>F</td>
<td>3</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>Negative control</td>
</tr>
<tr>
<td>60</td>
<td>G</td>
<td>4</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>Negative control</td>
</tr>
<tr>
<td>70</td>
<td>F</td>
<td>4</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>Negative control</td>
</tr>
<tr>
<td>54</td>
<td>F</td>
<td>3</td>
<td>50,000</td>
<td>154</td>
<td>46</td>
<td>2 degenerated</td>
</tr>
<tr>
<td>55</td>
<td>F</td>
<td>4</td>
<td>50,000</td>
<td>156</td>
<td>9</td>
<td>2 degenerated</td>
</tr>
<tr>
<td>63</td>
<td>F</td>
<td>3</td>
<td>100,000</td>
<td>155</td>
<td>290</td>
<td>27 degenerated</td>
</tr>
<tr>
<td>65</td>
<td>F</td>
<td>6</td>
<td>100,000</td>
<td>154</td>
<td>16</td>
<td>3 degenerated</td>
</tr>
<tr>
<td>7433</td>
<td>F</td>
<td>4</td>
<td>100,000</td>
<td>154</td>
<td>82</td>
<td>3 degenerated</td>
</tr>
<tr>
<td>53</td>
<td>F</td>
<td>4</td>
<td>150,000</td>
<td>160</td>
<td>341</td>
<td>274 degenerated</td>
</tr>
<tr>
<td>61</td>
<td>G</td>
<td>3</td>
<td>150,000</td>
<td>155</td>
<td>502</td>
<td>Used as negative control</td>
</tr>
<tr>
<td>68</td>
<td>F</td>
<td>5</td>
<td>150,000</td>
<td>160</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

F indicates Friesian
G indicates Guernsey
P.M. represents postmortem.

Sera

These were obtained from the 7 experimental calves (Table 1) and also from 285 adult slaughter cattle at the Kenya Meat Commission (KMC) abattoir, Athi River. Postmortem findings on the slaughter cattle are given in Table 2. One hundred and ninety-five animals had parasites and/or gross pathological lesions while 90 were apparently "normal" cattle. The 285 sera constituted batch A in this work. Another batch of sera, referred to as batch B, was also obtained from adult slaughter cattle at the KMC abattoir. Details on postmortem findings of the animals from which batch B was collected are summarized in Table 3.

Table 2: Postmortem findings* on slaughter cattle from which batch A of sera were collected.

<table>
<thead>
<tr>
<th>No. of sera</th>
<th>Postmortem findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>39</td>
<td>C. bovis (&gt; 5 cysts)</td>
</tr>
<tr>
<td>20</td>
<td>C. bovis (&lt; 5 cysts)</td>
</tr>
<tr>
<td>47</td>
<td>E. granulosus only</td>
</tr>
<tr>
<td>41</td>
<td>Fasciola spp. only</td>
</tr>
<tr>
<td>18</td>
<td>Calcified parasitic lesions in liver only</td>
</tr>
<tr>
<td>8</td>
<td>Lung oedema only</td>
</tr>
<tr>
<td>7</td>
<td>Lung emphysema only</td>
</tr>
<tr>
<td>1</td>
<td>Lung abscess only</td>
</tr>
<tr>
<td>1</td>
<td>Lungworm lesions only</td>
</tr>
<tr>
<td>3</td>
<td>Perihepatitis only</td>
</tr>
<tr>
<td>7</td>
<td>Liver abscess only</td>
</tr>
<tr>
<td>3</td>
<td>Peritonitis</td>
</tr>
<tr>
<td>90</td>
<td>No parasites or gross pathological lesions</td>
</tr>
</tbody>
</table>

* Observed at routine meat inspection.

Antigens

Oncospheres artificially hatched and activated in vitro (Silverman, 1954a) were used in the IFA test. A crude T. saginata extract and a partially purified fraction designated FI (i.e. the first protein fraction obtained by Sephadex G-200 filtration) were used in the IHA test. The methodology for the preparation of the antigens was as given by Gathuma (1977).
Table 3: Postmortem findings* on slaughter cattle from which batch B of sera were collected

<table>
<thead>
<tr>
<th>No. of sera</th>
<th>Postmortem findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>C. bovis + E. granulosus</td>
</tr>
<tr>
<td>20</td>
<td>C. bovis + Fasciola spp</td>
</tr>
<tr>
<td>12</td>
<td>C. bovis + calcified parasitic lesions in liver</td>
</tr>
<tr>
<td>62</td>
<td>Mixed infections**</td>
</tr>
</tbody>
</table>

* Observed at routine meat inspection.
** Mixed infections refer to infection with C. bovis and at least 2 of the following: E. granulosus, Fasciola spp., calcified parasitic lesions in the liver.

Preparation of gelatin coated slides and fixing of oncospheres for the IFA test

Slides (75mm x 25mm) were thoroughly cleaned, wiped with cotton soaked in alcohol and air-dried. Several drops of 1% gelatin solution were added to the centre of each slide and the gelatin dried for several hours or overnight at 40°C.

Activated oncospheres were fixed with 10% formol-saline for 10 minutes, washed in two changes of distilled water and finally resuspended in approximately 1 ml of distilled water. One drop of the oncosphere suspension was placed on the gelatin layer on the slides and dried for several hours or overnight at 40°C. Prepared slides were either used immediately or stored at −20°C until required.

Procedure for the IFA test.

Test sera were inactivated at 56°C for 30 minutes and absorbed with an equal volume of packed sheep red blood cells at room temperature for 30 minutes. They were then centrifuged at 5,000 x g for 10 minutes and the sera recovered. Serial doubling dilutions of test sera in PBS, pH 7.2 were made in V-bottomed microtitre plates using 0.025 ml of PBS, pH 7.2 as diluent. An equal volume of cells was added to each well and left to stand at room temperature. For each test, a known positive serum, a known negative serum, nonsensitized cells and controls without serum were included. Results were read after 1 and 3 hours.

Procedure for the IFA test.

The gelatin coated slides with T. saginata oncospheres embedded in gelatin were rinsed in distilled water. Test sera were inactivated at 56°C for 30 minutes. Serial doubling dilutions of sera were made in PBS, pH 7.4. Several drops of each serum dilution were placed on each slide and the slides left at room temperature for 30 minutes with occasional shaking.

The slides were then washed with PBS, pH 7.4 to remove excess sera. They were subsequently soaked for 10 minutes in each of two changes of PBS, pH 7.4 The slides were dried and one drop of 1:32 dilution of fluorescent reagent (fluorescein conjugated rabbit antiovine globulin globulin) was added. The use of this particular dilution was arrived at after block titration with known positive and negative sera.

The slides were incubated for 30 minutes at 37°C in a moist chamber, rinsed with PBS, pH 7.4 and then soaked for 10 minutes in each of two changes of PBS, pH 7.4. They were blotted dry, mounted with buffered glycerine (10% PBS, pH 7.4, in glycerol) and examined with a fluorescent microscope (Ernst Leitz G.M.G.H. Wetzlar, Germany). Specific fluorescence occurred in the cells of the hexacanth embryo.
Results of the IHA and IFA tests were compared using the t-test.

Mean geometric titres of IHA and IFA tests were compared using 311 sera of slaughter cattle, 221 of which had either single or multiple infections and 90 non-infected controls.

**RESULTS**

Figures 1 and 2 show comparisons of the mean titres and standard errors of the IFA and the IHA tests using crude antigen and fraction FI antigen, respectively, on sera of experimentally infected calves.

The figures reveal that the mean IFA titres were slightly higher than the mean IHA titres using either crude or FI antigen.

The four negative control calves had IFA titres ranging from 1:4 to 1:32. All sera tested showed some fluorescence at day 0 post-infection, but significant titres (> 1:64) appeared 7 days post-infection. In the IHA test, most calves had detectable antibody response 2–4 weeks post-infection. The four negative control calves either showed no agglutination or showed titres of 1:2.

Table 4 summarizes the results of the IHA test using crude and FI antigens and IFA test on sera of 285 adult slaughter cattle. Table 5 shows the geometric mean IFA, IHA (crude) and IHA (FI) titres on sera of 311 adult slaughter cattle, 221 of which had various infections and 90 “normal” cattle. When the mean IFA titres were compared with the mean crude antigen IHA titres of sera of 47 cattle infected with *E. granulosus* (hydatid cysts) only, the latter were significantly higher (P < 0.01). There was no significant difference between these tests using sera from 59 cattle infected with *C. bovis* only, 21 cattle infected with *C. bovis* and *E. granulosus* and 20 animals infected with *C. bovis* and *Fasciola* spp. There was also no significant difference between the two

![Graph](image-url)

**Fig. 1. Mean titres and standard errors of IFA and IHA (crude antigen) tests (n = 7)**
Table 4: Results of IHA test using crude and FI antigen, and IFA test using activated oncospheres as antigen.

<table>
<thead>
<tr>
<th>Post-mortem findings*</th>
<th>No. tested</th>
<th>Percentage &quot;positive&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>IHA (crude)</td>
</tr>
<tr>
<td><em>C. bovis (&gt; 5 cysts)</em></td>
<td>39</td>
<td>87)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>71.2</td>
</tr>
<tr>
<td><em>C. bovis (&lt; 5 cysts)</em></td>
<td>20</td>
<td>40)</td>
</tr>
<tr>
<td><em>E. granulosus only</em></td>
<td>47</td>
<td>59.6</td>
</tr>
<tr>
<td><em>Fasciola spp. only</em></td>
<td>41</td>
<td>2.4</td>
</tr>
<tr>
<td>Calcified parasitic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lesions in liver only</td>
<td>18</td>
<td>22.2</td>
</tr>
<tr>
<td>Lung oedema only</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Lung emphysema only</td>
<td>7</td>
<td>14.3</td>
</tr>
<tr>
<td>Lung abscess only</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Lungworm lesions only</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Perihepatitis only</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Liver abscess only</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>No parasites or gross</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pathological lesions</td>
<td>90</td>
<td>11.1</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>285</td>
<td></td>
</tr>
</tbody>
</table>

* Observed at routine meat inspection.
Table 5: Geometric mean titres of sera from animals with various parasitic infections

<table>
<thead>
<tr>
<th>Post-mortem findings*</th>
<th>No. tested</th>
<th>Mean (geometric) titres</th>
<th>IHA (Crude)</th>
<th>IHA (FI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>IFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. bovis only</td>
<td>59</td>
<td>8.92 (512)**</td>
<td>5.90 (64)</td>
<td>7.68 (25)</td>
</tr>
<tr>
<td>E. granulosus only</td>
<td>47</td>
<td>1.30 (2)</td>
<td>5.81 (64)</td>
<td>4.60 (32)</td>
</tr>
<tr>
<td>C. bovis + E. granulosus</td>
<td>21</td>
<td>4.00 (16)</td>
<td>9.05 (512)</td>
<td>4.52 (32)</td>
</tr>
<tr>
<td>C. bovis + Fasciola spp.</td>
<td>20</td>
<td>3.95 (16)</td>
<td>8.75 (512)</td>
<td>3.20 (8)</td>
</tr>
<tr>
<td>C. bovis + calcified parasitic lesions in liver</td>
<td>12</td>
<td>5.00 (32)</td>
<td>5.50 (64)</td>
<td>5.08 (32)</td>
</tr>
<tr>
<td>Mixed infections***</td>
<td>62</td>
<td>4.35 (16)</td>
<td>8.37 (256)</td>
<td>4.53 (32)</td>
</tr>
<tr>
<td>No parasites</td>
<td>90</td>
<td>1.29 (2)</td>
<td>1.98 (4)</td>
<td>1.14 (2)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>311</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* observed at routine meat inspection.
**Figures in parentheses represent approximate mean reciprocal titres.
***Mixed infections refer to infection with C. bovis and at least 2 of the following: E. granulosis, Fasciola spp., calcified parasitic lesions in the liver.

tests on sera from 12 cattle infected with C. bovis and also having calcified parasitic lesions in the liver or 90 “normal” control cattle.

Comparison of the mean IHA titres using FI antigen and IFA test by t-test for correlated data revealed similar results to those obtained by comparing mean IHA titres using crude antigen and mean IFA titres. The mean IHA titre using FI antigen on sera of 47 cattle infected with E. granulosus was significantly higher than the mean IFA titre of the same animals (P < 0.05). The mean IHA titre using FI antigen and the mean IFA titre on sera of 59 cattle infected with C. bovis only did not differ significantly. Likewise, there was no significant difference between the IHA titre using FI antigen and IFA titres on sera of 21 cattle infected with C. bovis and E. granulosus only, 21 cattle infected with C. bovis and Fasciola spp. only, 12 cattle infected with C. bovis and also having calcified parasitic lesions in the liver only, 62 cattle with mixed infections or 90 “normal” control cattle.

Evaluation of sensitivity and specificity

Of 59 cattle found to have C. bovis only at post-mortem examination, 54 produced sera which gave significant titres (> 1:64) with the IFA test, thus giving a sensitivity of 91.5%. Forty-two of the 59 sera showed significant titres in the IHA test using crude antigen (71.2% sensitivity). When the two sensitivity proportions were compared by t-test for correlated proportions, it was found that the sensitivity of the IFA test was significantly higher than that of the IHA test using crude antigen (P < 0.01). Of the 226 sera from cattle found to have no C. bovis infection at postmortem inspection, 204 sera did not show significant titres in the IFA test. This gives a specificity of 90.2%. Sera from 182 of the 226 C. bovis negative cattle did not show significant titres in the IHA test using crude antigen. This means the specificity of this
test was 80.5%. Comparison of the two specificity proportions by t-test revealed that the IFA test was more specific than the IHA test using crude antigen (P < 0.01).

Results of the IHA test using FI antigen showed a sensitivity of 88.1% (52 out of the 59 C. bovis positive cattle showed significant titres) and a specificity of 87.6% (198 of the 226 C. bovis negative cattle did not show significant titres). These sensitivity and specificity values did not differ significantly from those of the IFA test.

DISCUSSION

The IFA test using activated oncospheres as antigen gave significantly higher mean titres with sera of experimentally infected calves than the IHA test using either crude antigen or fraction FI. This confirms the observations of Machnicka (1973, 1974) who obtained higher titres with the IFA test using activated oncospheres than with the IHA test although Machnicka (1973, 1974) used C. bovis cyst fluid as antigen. With sera of adult slaughter cattle, the IFA test was as sensitive as the IHA test using FI antigen, but was more sensitive than the IHA test using crude antigen. Although the specificity of the IFA test was significantly higher than the specificity of the IHA test using crude antigen, it did not differ significantly from that of the IHA test using FI antigen. The IFA test gave a lower rate of cross-reactivity with sera of animals infected with E. granulosus (10.6%) than the IHA test using crude antigen (59.6%) or FI antigen (31.9%). This means that, with regard to E. granulosus-infected animals, the IFA test was more specific than the IHA test. Using cryostat sections of proglottides of T. solium in the IFA test, Daoe et al. (1972) found the IFA test more specific than the IHA test. Rydzewski et al. (1975) reported similar results in the IFA test using paraffin sections of T. saginata in the diagnosis of human cysticercosis.

There seems to be general agreement, therefore, that the IFA test is more sensitive and more specific than the IHA test. Nevertheless, the IFA test, in our experience in this study showed some drawbacks which make it less useful as a diagnostic or screening test in bovine cysticercosis. Firstly, non-specific fluorescence was observed especially in cases where the oncospheres had not been completely freed from the oncosporal membrane. Machnicka (1973, 1974) obtained similar results. This necessitated the use of only fresh and mature T. saginata eggs to ensure good hatchability.

Silverman (1954b) pointed out the uneven fertility of ova within proglottides of T. saginata. According to him, the gravid proglottides of T. saginata and T. pisiformis contain 50% mature ova, 40% immature ova and 10% infertile ova. Mature ova in varying degrees (0—80%), are present only in the distal 30-50 proglottides. Obtaining a batch of mature and fertile ova with good hatchability is, therefore, a problem which may severely restrict the routine use of this test. Secondly, although the IFA tests are simple to perform and are easily standardized, there are several potential problems in their application. Results are liable to subjective interpretation and must always be related to the reactive and non-reactive controls.

ACKNOWLEDGEMENT

This study was supported by a grant from the Rockefeller Foundation awarded to the Faculty of Veterinary Medi-
cine, Kabete, for research on cysticercosis. This financial assistance is gratefully acknowledged.

REFERENCES


Received for publication on 19th October, 1979
PRIMARY AND SECONDARY HUMORAL IMMUNE RESPONSES IN CATTLE EXPERIMENTALLY INFECTED WITH DERMATOPHILUS CONGOLENSIS

A. A. MAKINDE and A.O. EZEH
National Veterinary Research Institute, Vom, Plateau State, Nigeria.

SUMMARY

Four cattle were experimentally infected twice with Dermatophilus congoensis and the immune responses monitored every 5 days for a period of 100 days. The primary infection was allowed to run its course and after a period of 65 days post infection a second infection was carried out on the same animals and the immune responses again monitored for the next 35 days before the experiment was stopped.

An anamnestic response was recorded, which was much higher than the primary one and this corresponded to the time of the clearing of the lesion in the animals.

INTRODUCTION

Antibodies to Dermatophilus congoensis have not been known to influence the development of the lesions of streptothricosis or to prevent their formation in previously infected animals, though they are often detected as early as 5 days after infection (Makinde, 1979). Animals in various stages of infection with D. congoensis have often been examined for antibodies but the levels never corresponded with the severity of such infections (Merkal, Richard, Thurston and Ness, 1972; Pulliam, Kelley and Coles, 1967).

It is, therefore, necessary to examine primary and secondary immune responses in experimentally infected animals in order to relate the responses to what is expected in natural infections where case histories of the disease are often either inaccurate or non-existent.

MATERIALS AND METHODS

Animals

Four Zebu heifers (White Fulani) aged 9 – 12 months were used. They were dewormed with Thiabendazole (Merk, Sharp & Dohme Ltd., England) two weeks before the start of the experiment while daily rectal temperature-taking was begun a week after deworming. The animals were kept together in a non-fly proof, pen. They were in good physical condition and had adequate feeds.

Culture of D. congoensis

A strain of D. congoensis obtained from a chronically infected cow that died in extremis was used for infection. The organism was grown in trypticase soy broth (BBL, Maryland, USA) for 72 hours and concentrated by centrifugation at 500 x g for 30 minutes.

Scabs obtained from lesions produced by the experimental infection were treated according to the method described by Haalstra (1965) for the isolation of D. congoensis. Such organism was also confirmed by positive reaction to Giemsa staining.

Infection of Animals

Two areas (10 x 10 cm) on either side of the dorsal region of each animal were shaved and scarified with a specially-designed nail-brush. One area had 20 ml of concentrated suspension of broth culture of D. congoensis applied to it while the other area had ordinary trypticase soybroth applied to it to serve as control.
All the animals were bled before infection and every 5 days for 35 days after infection. After 30 days rest, i.e. 63 days after the first infection, all the animals were bled and re-infection carried out as described above and again bleeding was carried out every 5 days until the termination of the experiment after 100 days.

All the animals were observed in order to monitor the development of the lesions. All the serum samples collected during the course of the experiment were kept at $-20^\circ$C until they were subjected to serological tests after the completion of the experiment.

**Preparation of *D. congoensis* antigens**

**Ammonium sulphate extraction method**

Extracellular extracts of the broth culture were prepared by ammonium sulphate extraction. The broth culture was centrifuged at 500 x g for 30 minutes to remove the sediment and the supernatant was filtered through a 0.22μm filter (Millipore S.A. Buc-France). The cell-free filtrate was treated with ammonium sulphate (80% saturation) and left overnight at 4$^\circ$C for precipitation of proteins. The precipitate was removed by centrifugation at 15,000 x g for 30 minutes at 4$^\circ$C dissolved in 5 ml of cold 0.15 M NaCl, and dialysed against distilled water for 3 days with frequent water changes to remove ammonium sulphate ($\text{NH}_4\text{SO}_4$) before it was finally lyophilized.

**Trichloroacetic acid (TCA) extraction method**

10 mg of each moist culture sediment obtained from (a) was suspended in 25 ml of ice-cold trichloroacetic acid (TCA) and processed as described by Lanyi, Adam, Szentmihalyi (1975). The suspension was left to stand at 4$^\circ$C for 24 hours after which it was centrifuged at 10,000 x g for 30 minutes. The supernatant was collected and kept at 4$^\circ$C, and the sediment resuspended in another 25 ml of 10% TCA for another 24 hours. After the second extraction the supernatants were pooled and dialysed against distilled water for 3 days at 4$^\circ$C with frequent water changes. The dialysate was then lyophilized.

**Passive Haemagglutination Test**

Based on the method described by Gold and Fudenberg (1967) and as modified by Godding (1976), the test was carried out using sheep red blood cells (SRBC) sensitized with Chromium Chloride (CrCl$_3$).

The sera to be tested were first heat-inactivated at 56$^\circ$C for 30 minutes and absorbed with washed SRBC. The plate test was employed using Cook Micro titre (R) System.

Serial two-fold dilutions of the test sera were made in the first row of twelve wells with 0.05 ml PBS in each well up to well 10. Wells 11 and 12 contained 0.05 ml test serum. A 1% sensitized SRBC was added in 0.05 ml amounts from wells 1 to 11 and 0.05 ml PBS was added to well 12 instead. Another row of twelve wells was similarly treated using a known negative serum. The contents of each well were mixed and the results read after incubation at room temperature for two hours. The pattern of settling of the cells was carefully recorded. A tight button of the cells was recorded as negative. A positive varied from a thin carpet of cells covering the entire bottom of the well to an agglutinated pattern surrounded by an irregular ring.
RESULTS

Infection

Generalized inflammatory reactions were observed on all the scarified sites 24 hours post-infection but these subsided in control sites while the infected sites became hyperaemic and papular at 48 hours. In 5-8 days scab formation was observed on the infected sites and by day 10 the scabs coalesced to form crusts which were difficult to remove. The crusts obtained from day 10 to day 25 were positive for *D. congolensis* in culture and by Giemsa staining. No organism could be cultured from the control sites at any time. By day 25 all the lesions had healed in 3 animals while a fourth animal 737 which still possessed scabs continued to be positive for the presence of *D. congolensis* until the termination of the experiment, though its lesions were limited to the area scarified. The response to the second infection was similar but lesions cleared after 10 days except for animal 737.

Antibody titration

A definite pattern of antibody responses to *D. congolensis* extract was established during the course of the infections as antibodies were found at lower levels in the primary infection than in the secondary infection.

The TCA extract detected higher antibody responses during the infections and also stimulated earlier responses than the (NH₄)₂SO₄ extract. (See Figure 1).

DISCUSSION

Experimental infections of animals with *D. congolensis* have yielded several observations particularly with respect to the course of such infections and the role of antibody in the termination of the infection. Macadam (1964) observed that experimentally-induced lesions of streptothricosis lasted 15 days in cattle, 18 days in ponies, 42 days in goats and 30 days in sheep while Abu-Samra and Imbabi (1976) found that in all species he infected experimentally the lesions healed completely within 20-25 days. Makinde (1979) also found that complete healing took place in rabbits between 18 to 22 days post-infection. Buggyaki (1959), however, noted that recovery from streptothricosis does not appear to give rise to immunity to other attacks.

This study shows similar results with respect to the healing of lesions (20-25 days for first infection and 10-15 days for second infection) which incidentally coincided with the periods the serum antibody titres were relatively high. The rapid healing which was observed during the second infection indicated an anamnestic response. In this respect the observation made by Macadam (1964) that rapid healing usually followed a second scarification and that by Robert (1966) that vaccination increased by 16-fold the resistance of scarified skin in sheep and guinea pigs which he associated with circulating somatic antibody, are pertinent to this study.

The presence of high antibody levels during a second infection is noteworthy since this is likely to be responsible for the shorter duration of the infection. However, TCA extract, induced an earlier and a higher antibody responses than those induced by the (NH₄)₂SO₄ extract. Makinde (1979) also observed an early antibody response to “whole cell antigen” and “endoantigen” which are similar to the TCA extracts.

It is, therefore, apparent from the results of this study that difficulties
Fig. 1: Mean serum haemagglutinating antibody titres specific for Ammonium Sulphate and Trichloroacetic Acid extracts of *D. congolensis*. 

*Received for publication on 1st April, 1980*
ENDOPARASITES IN DOMESTIC AND WILD ANIMALS OF THE CENTRAL-AFRICAN REPUBLIC (C A R)

M. GRABER,
Head of Parasitology Department, Ecole Nationale Vétérinaire de Lyon, Marcy L'Etoile, 69260 Charbonniers, Les Bains, France.

SUMMARY

Between 1959 and 1972 an unestimated number of domestic and wild animals (carnivores, Artiodactyloids, primates, birds, amphibians and reptiles) were autopsied in the animal husbandry region in the West of the Cygenetic Area and to the west of the Centrafrican Republic.

Two hundred and twenty nine different parasites were identified as belonging to the following groups; Trematoda, 32; Cestoda, 42; Nematoda, 141; Acantocephala, 1; Poroccephala, 2 and Myiasis agents, 11. 46 new species and one sub-species were also identified, as well as a particular lion adapted Echinococcus granulosis granulosus stock.

On the whole, parasitism in wild animals is very low. It is well tolerated except in buffaloes and bush pigs where there appears sometimes to be a marked decline in general state.

Parasitism in domestic ruminants is much more serious, especially in juvenile veals suffering from ascaridiosis and strongyloidosis, in steers infected by digestive strongylosis, in sheep and in poultry whose raising is hampered by the presence of numerous endoparasites.

Irrespective of the country to which they belong, the wild animals of the extensive African fauna show a remarkable homogeneity and, except in very few cases, the same parasites are found, be it in CAR, Chad, East Africa (Elizabeth National Park, Serengeti Park, Kenya Parks) in Central Africa (Zaire) and in South Africa (Kruger Park).

INTRODUCTION

The Central African Republic is situated in the heart of Central Africa. It stretches from Chad Republic in the north (Latitude 11° north) to Zaire (4th parallel) and the Peoples Republic of Congo (3rd Parallel) in the south. It is bordered on the east by Sudan (27° meridian) and on the west by the Federal Republic of Cameroon (15° Meridian).

The area between latitude 7½° and 11° north forms the southern zone of the Chadian territory. This a relatively flat area strewn with some hills or plateaux. In addition the relief is formed by huge mountain ranges which include the Yade in the West and the Bongos in the East. Its average altitude varies between 900 and 1,200 metres. It stretches southwards and eastwards by a range of low altitude plateaux. This granitic barrier marks the water sharing line between the Chari basin (Lim, Pende Bahr Sara) in the West; Bahr Aouk in the East) and the Zaire basin (Sangha, Lobaye, Kotto, M'bomou).

The climate is generally tropical humid characterized by a single rainy season during the northern summer. From the 7th to the 11th parallel the climate is the sahelo-Sudanese type with four months marked rainy season and an annual rainfall of 900 to 1,300 mm. From the 3rd to the 7th parallel the climate is the Sudano-Guinean type. The rainy season lasts six months and precipitation height ranges from 1,300 to 1,600 mm.
The former area is covered by clear savannah shrubs and trees intersected with large strips of herbaceous pasture grounds and the latter by thick forest galleries along the waterways.

Animal raising in the Central African Republic is a recent creation. It was in 1924 that the first Bororo herdsmen occupied the Yade savannah for the first time. Since then they have scattered into the entire west of the country (Bouar, Baboua, Haute Sangha, Ouham Pande) as well as into the east (Bambari). In addition there exists some trypanotolerant bovine flocks (Ndamas, Baoule) in the glossina-infested areas.

Currently the size of bovine live stock in the area is estimated at roughly 800,000. Goat and poultry raising is equally flourishing. Wildlife constitutes the second wealth of C A R. It occupies in the east of the country an immense area of 320,000 Km². Its limits are shown on the map. Its synegetic capital is one of the richest in Africa, the most abundantly represented species being the buffalo (300,000 heads) the elephant (20,000 heads) the wharthog and various antelopes, in particular the bubal (Thal, 1972). Both domestic and wild animals are subject to a number of infections among which endoparasites, owing to the particularly favourable climatic conditions, occupy an important place. It has therefore been interesting to know their agents.

MATERIALS AND METHODS

Between 1959 and 1972 three series of investigations were conducted and a large number of animals were sacrificed and autopsied.

(1) Perissodactylous, artiodaclylous and domestic carnivorous -- poultry. The autopsies were performed in slaughter houses in Bouar, Bangui and Bambari. They were performed on 3 asses, 10 pigs, 844 cows, 38 sheep, 4 goats, 2 dogs and 137 fowls.

(2) Artiodaclylous and wild carnivorous, proboscidea.

A large number of endoparasites were collected during the implementation of the FAO/UNDP/CAF 13 project (Thal 1972). On this occasion investigations were conducted on 208 animals including 74 wharthogs, 2 bush pigs 3 Giant Foresthogs, 74 African buffaloes, 3 Derby Eland, 4 Bush-bucks, 14 Bubals 1 Damalisc, 7 Hippotragus 7 water bucks 5 Cob de buffalo 3 Crowned common duckers, 5 oribis, 4 Elephants 1 Civet Cat, 1 inchneumon mongoose 2 Hyenas 3 lions and one panther. In the case of primates, insectivores, rodents and amphibians the parasites were collected in Maboke Station and its immediate neighbourhood (Boukoko, Bebe). In general, cattle were imported from Chad and from the experimental station of the natural history museum of Paris, situated in South West Bangu, M’baiki region (Lobaye).

Where reference is not being made to largely represented parasites the collection Stations are indicated by the coordinate I.B.A.R., which uses squares divided into longitudes and latitudes.

The list prepared also includes Myiasis and porocephalidae agents which may be considered in broad terms as endoparasites whose action adds to the action of other helminths.
PARASITES ENCOUNTERED

1–1– Trematoda
1–1– Monogenea

Polystomatidae Family Gamble, 1896
Host: Hylarana albolarbis
Location: Bladder
Collection Station: La Maboke (Euzet et al, 1974)
Polystoma llwellyni (Euzet, Combes and Knoepffler, 1974)
Host: Africathalas fulvoventitius.
Location: Bladder
Collection Station: La Maboke
Polystoma prudhoei Saoud, 1867
Host: Ptychadena oxyrhynchos
Location: Bladder.
Collection Station: La Maboke (Euzet et al., 1974)
1–1–2– Digenea.
1–1–2–1– Dicrocoelidae family odhaer 1911 Dicrocoelium hospes Looss, 1907
Host and infestation rate: Zebu (27%) Buffalo (10%).
Collection station: Bouar (Graber and Oumetie) 1964;
Graber et al, (1969) the entire cymegetic area (Graber and Thal, 1979 b)
1–2–2– Fasciolidae family Railliet 18925.
Fasciola gigantica Cobb. Cox, 1855.
Host and infestation rate: zebu (62%)
Sheep, buffalo (37%); Bubal (7%);
Cob de buffon; Water buck (55%)
Hippotragus, what-hog (1.5%)
Location: hepatic parenchyma and bile ducts.
Collection stations: Bouar, Carnot, Bangui, Bambari, Bira and the entire Cymegetic area, from the 5th to the 9th parallel.
(Graber and et al.1969; Troncy et al., 1973a;
Graber and Thal, 1979 b)
Protofasciola Robusta von Lorenz, 1881
Host: Elephant (one out of four)
Location: intestine
Collection Station 24–6–B.d
Brachylaima attenuatum Baeer, 1933.
Hosts: A little bearded yellow monstachened bulb.
Location: Intestine
Collection station: CAR (Fischthal, 1977)
1–2–4– Plagiorhidae family Patt, 1902
Maederia eburnense maeder, 1969
Host: Hylarana albolarbis albolarbis
Location: duodenum
Collection station — La Maboke (Maeder et al., 1970)
1–2–5– Brachycodiella family Johnston, 1912.
Mesocoelium Monas (Rudolphi, 1810 Freitas 1958)
Host: Hylarana labolarbis, albolarbis ptychadena; Oxyrhynchus; Ptychadena perreti;
Ptychadena superciliasis; chiromantis refescaus.
Location: duodenum
Collection station: La Maboke (Maeder et al, 1970)
Mesocoelium gabonensis Maeder, Combes and Knoepffler, 1969
Hosts: Ptychadena superciliasis;
Ptychadena Perreti; Ptychadena Oxyrhynchus
1–2–6– Schistosomatidae family poche, 1907.
Schistosoma bovis Sonsino, 1876
Host: Zebu
Location: Hepatic and Mesentric Veins.
Collection Station: Bangui. The Schistosomosis bovis is practically unknown in the east and in the west of CAR. It can only be seen in Bangui among herds of Chadian origin (Graber, 1961; Graber et al, 1969).
1–2–3– Paramphistomatidae family Fischhoeuder, 1901.

Hosts and infestation rates: Zebu (2%)
Buffalo (20%) Cob de buffon; Hippotragus water buck and oribi.
Location: Gastric reservoirs
Paramphistomum clavula Nasmack, 1937
Hosts and infestation rates: Buffalo (8%) Hippotragus;
Location: Gastric reservoirs
Paramphistomum phillouxi Dinnik, 1961
Hosts and infestation rates: Buffalo (2.7%)
Cob de buffon; water buck and Hippotragus
Location: Gastric reservoirs
Collection station: 21.9.b; 23.7.c.d; 23.7.
A. d; 23.9.c.c; 25.5.c.c. (Sey and Graber, 1979 a and Graber and Thal, 1981.)

**Bothriophoron bothriophoron** (braun, 1892)

*Fischoeder, 1901*

**Host:** Buffalo (1%)

**Location:** Gastric reservoirs

**Collection station:** 21.8. c.d. (Graber and Thal, 1981).

**Gigantocotyle symmeri** Nasmak, 1937

**Hosts:** Buffalo (2.7%) Hippotragus; Bubal

**Location:** Gastric reservoirs


**Cotyphorhonus macrospheictris** Sey and Graber, 1979

**Location:** Gastric reservoirs

**Collection stations:** The entire cynegetic area of eastern CAR, from the 5th to the 10th parallel (Sey and Graber, 1979; Graber and Thal, 1981).

**Cotyphorhonus macrospheictris** Sey and Graber, 1979

**Hosts and infestation rates:** Buffalo (70%); Bubal (21%)

**Location:** Gastric Reservoirs

**Collection stations:** The entire cynegetic area of eastern CAR from the 5th to the 10th parallel (Sey and Graber, 1979 b; Graber and Thal, 1981).

**Cotyphorhonus cotyphororum** Fischoeder, 1901.

**Hosts and infestation rates:** Zebu (86%); Sheep, goat, Bubal (70%); Oribi; Bush-buck water buck; Damalisic.

**Location:** Gastric reservoirs

**Collection stations:** Paoua, Bouar, Carnot, Bambari, the entire cynegetic area of Eastern CAR; (DOUFUS, 1950; Farcha yearly report, 1968; Graber and Thal, 1981).

1—2—5—) **Gastrothylacidae** Family stiles and Goldberger, 1910.

**Carmeryrius endopapillatus** Doufus, 1962

**Host:** Buffalo (4%)

**Location:** Gastric reservoirs


**Carmeryrius exporus** mapstone, 1923

**Hosts:** Buffalo and Hipotragus. Rarely met tremetoda

**Location:** Gastric reservoirs


**Carmeryrius gruberi** Gretillat, 1960

**Hosts and infestation rates:** Zebu (3.6%); Buffalo (8%) Bubal 7%;) Bushbuck and Reed-buck

**Location:** Gastric Reservoirs

**Collection stations:** Bouar, Cynegetic area from 5th to 9th parallel (Forcha yearly report 1968; Graber et al, 1969; Sey and Graber, 1979 a);

**Carmeryrius gregarius** Looss, 1896

**Host:** Buffalo (only once)

**Location:** Rumen

**Collection Station:** 20.7 c.b (Graber and Thal, 1981)

**Carmeryrius minutus** Fischoeder, 1901.

**Host:** Bubal (Once)

**Location:** Gastric Reservoirs

**Collection Station:** 22.9 c.a. (Graber and Thal, 1981).

**Carmeryrius papillatus** Gnetillat, 1962

**Hosts and infestation rates:** Zebu (1.2%); Buffalo (10%) Water buck; Hippotragus, Bubal.

**Location:** Gastric Reservoirs

**Collection stations:** Bouar, Bangui and the Cynegetic area from the 6th to the 9th parallel (Graber, 1961; Graber et al., 1969; Graber and Thal, 1981).

**Carmeryrius Parvipapillatus** Gnetillat, 1962

**Host:** Zebu (rarissime)

**Location:** Gastric Reservoirs

**Collection station:** Bouar (Graber et al., 1969)

**Carmeryrius Schoutedeni** Gnetillat, 1962

**Host:** Water buck and Reedbuck (rare)

**Location:** Gastric Reservoirs

**Collection Station:** 17.10. Ac (Graber and Thal, 1981).

**Carmeryrius spatius** Brandes, 1899

**Hosts and infestation rates:** Zebu (0.7%); Buffalo (24%); Drby Eland, Water buck; Cob de buffalo; bushbuck, bubal and Hippotragus (one each of every two)

**Location:** Gastric Reservoirs

**Collection Stations:** Bouar, Bangui and the entire cynegetic area in eastern CAR (Graber 1961, Farcha yearly report, 1968) Graber et al., 1969).

**Carmeryrius Spatius** is the best represented carmeryrius in CAR. Generally, (Graber and Thal, 1981), Carmeryrius are much more in abundance among wild bovine than among domestic bovine (43% as against 4% of animals
slaughtered) whereas in the case of Paramphistomatidae infestation rates are almost similar (87% and 89%).

1—2—9—) *Stephano pharyngidae* family skrjabin, 1949.

*Stephano pharynx Coilos* Dollfus, 1963
Hosts: Buffalo, Water buck, Cob de buffon and Hippopotagus. This is the first time the parasite is found in a buffalo (Sey and Graber, 1979 a)
Location: Gastric reservoirs

*Stephano pharynx comhactus* Fischeder 1901
Host and infestation rate: Buffalo (9.5%) Location: Gastric Reservoirs
Collection Station: Cynesetic area of 5th to 9th parallel (Sey and Graber, 1979 a Graber and Thal, 1981).

1—2—10— *Brumptiidae* Family skrjabin, 1949

Host and infestation rate: Elephant (4 out of 4 animals).
Location: intestine
1—2—11—) *Gastrodiscidae* family stiles and goldberger, 1910.

*Gastrodiscus aegyptiacus* cobbold, 1876
Hosts and infestation rate: Wharthog (50%)
Giant forest-hog (2 out of 3 cases)
Location: Large intestine and Caecum.
Collection station: Bouar and the entire cygnetic area of Eastern CAR (Graber and Coll; 1964; Troncy et al; 1972 a)

*Choerocotyle epuluensis* Baer, 1959
Hosts and infestation rates: Wharthog (one out of 48 cases) and Giant forest-hog (two cases out of three).
Location: Colon
Collection station: 25.5. D.d. (Troncy et al. 1972 a) 2 Cestoda.

2—1—) *Diphyllolobothriidae* family Luhe, 1910.

*Diphyllobothrium theileri* Baer, 1924
Host: Lion (One out of three)
Location: Small intestine

2—2—) *Anoplocephalidae* family, cholodkowsky, 1902.

*Catenotaenia lobata* Baer 1925
Host: Mastomys sp
Location: Intestine
Collection station: Boukoke; Bebe (Quentin, 1965 d and 1971 b).

*Skrjabinotaenia media* Quentin, 1971
Host: Bush mouse
Location: intestine
Collection Station: Maboke (Quentin, 1971b)

*Skrjabinotaenia gerbili wertheim* , 1954
Host: Dorsal band black rat; Bristled rat
Location: Intestine
Collection Station: La Maboke (Quentin 1971 b)

*Skrjabinotaenia baeri* Lynsdale, 1953
Host: tree dwelling rate
Location: intestine
Collection Station: Maboke (Quentine, 1971b)

*Moniezia expansa* Rudolph, 1810
Hosts and infestation rate: Zebu (less than 1%) sheep; goat; buffalo (one case out of 74)
Location: Intestine
Collection Station: Bouar, Bambari; 23.6 c.c. (Graber et al. 1969; Graber and Thal, 1979 c)

*Moniezia beneden* Moniez 1879
Hosts and infestation rates: Zebu (1%); sheep;
Location: Intestine
Collection Station: Bouar, Carnot (Graber, 1968; Graber et al; 1969)

*Moniezia mettami* Baylis, 1934
Host and infestation rate: Wharthog (35%)
Giant forest-hog (one out of 3 cases)
Location: intestine
Collection Station: Wharthog; the entire cygnetic area, Giant forest-hog 25.5. D.d. (Troncy and Colp, 1972 a).

*Bertiella studeri* Blanchard, 1891
Host: Monkey S P
Location: Intestine
Collection Station: Bouar.

*Theysaniezia ovilla* Rivolta, 1878
Host: Zebu (1.5%)
Location: Intestine
Collection Station: Bouar, Bangui (Graber, 1961 Graber et al; 1969)

*Crossotaenia baeri* Mahon, 1954
Host: Bush-buck
Location: bile ducts
Collection Station: Bouar (Graber et al; 1964)

*Avitellina centripunctata* Rivolta, 1874
Hosts and infestation rates: Sheep, Buffalo
(15%) Cob de buffon and crowned common duiker
Location: intestine
Collection Stations: Nouar and Cy genetic area of 5th to 9th parallel (Graber, 1961; Graber and Thal, 1979 c)

*Sitlesia hepatica* Wolffshugel, 1903
Hosts and infestation rates: Hipotragus (5 out of 7) Cob de Buffon (2 out of 5) water buck (7 out of 7)
Location: bile ducts
Collection Station: Cy genetic area from 6th to 9th parallel (Graber and Thal, 1979 c)

2—3—) *Davaineidae* Family Fuhrmann, 1907

*Eintermicapsifer madagascarensis*
Davaine, 1870
Hosts: tree-dwelling rat; Nile Rat; Red muzzled rat of Gabon
Location: Intestine
Collection Station: La Maboke, Bangui, Boukoko Bebe (Quentin 1964 c; Durette – Desset 1966)

*Eintermicapsifer congolensis* Mahon 1954
Host: Emin rat
Location: Intestine
Collection Station: Baiki (Wuetin, 1964 c)

*Porogynia paranai* Moniez, 1891 c)
Host: Guinea Fowl
Location: Intestine
Collection Station: Bouar

*Cotugnia meleagridis* Joyeux and Martin, 1936.
Host: Guinea Fowl
Location: Intestine
Collection Station: Bouar

*Raillietina (Raillietina) tetragona* Molin, 1858.
Host and infestation rate: Chicken (50%)
Location: Intestine
Collection Station: Bouar, Bangui, Bambari (Graber, 1961; Farcha report, 1968).

*Raillietina (Raillietina) echnobothrina* Mergin, 1881.
Host: Chicken
Location: Intestine
Collection Station: Bouar, Bangui and Bambari (Farcha Report, 1968).

*Raillietina (Raillietina) pintneri* Klapcoco 2, 1906.
Host: Guinea Fowl
Location: Intestine
Collection Station: Bouar.

*Raillietina (Raillietina) baeri* Meggitt and Subramanian, 1927.
Hosts: Mastomys S.P.; Jackson rat
Location: Intestine
Collection Stations: Bangui, Boukoko, Toukoulou (Wuetin, 1964 c)

*Raillietina (Paroniella) numida* Fuhrmann 1912
Host: Guinea Fowl
Location: Intestine
Collection Station: Bouar.

*Ascometra Numida* (Fuhrmann, 1909) Baer 1955
Host: Guinea Fowl
Location: Intestine
Collection Station: Bouar.

2—4—) *Dilepisidae* Family Railliet and Henry, 1909.

*Anomotaenia henii* Quentin, 1964
Host: Bristled rat
Location: Intestine
Collection Station: Boukoko (Quentin, 1964 c)

*Dilepis dollfusi* Wuetin, 1964
Host: Mastomys S.P.
Location: Intestine
Collection Station: Bebe (Quentin, 1964 c)

*Choanotaenia infundibulum* Block, 1979.
Host: Chicken
Location: Intestine
Collection Station: Bangui (Farcha report, 1968)

*Joyeuxella S.P.*
Host: Lion
Location: Intestine
Collection Station: 21.8. D.C.


*Hymenolepis petteri* Quentin, 1964
Host: Bristled rat
Location: Intestine
Collection Station: Boukoko (Quentin, 1964 c)

*Hymenolepsis diminuta* Rudolphi, 1819.
Hosts: Mastomys S.P.; Black Rat
Location: Intestine
Collection Stations: Toukoulou, Bangui (Quentin, 1964 c)

*Hymenolepsis carioca* Magalhaes, 1898
Host: Chicken
Location: Intestine
Collection Station: Bangui (Farcha yearly report 1968)

2—6—) *Taeniidae* Family Ludwig, 1886
Host: Spotted hyena (one out of 2 cases)
Location: Intestine
Collection Station: 21–8–D.d.
The corresponding Cysticercis infects the bubal, the hippotragus (21.8. D.C; 21.8. D.d;) and the buffalo (20.7. C.b.) It is found in the muscles (Grabber et al., 1973 b)

*Taenia gonyamai* Ortlepp, 1938
Host: Lion (one of three)
Location: Intestine
Collection Station: 21.8. D.d (Grabber et al, 1973 b)

*Taenia hyaenae* Baer, 1924
Host: Spotted hyena (two out of three cases)
Location: Intestine

*Taenia hydatigena* Pallas, 1766
Host: Dog
Location: Intestine
Collection Station: Bouar. The corresponding cysticercis, *Cysticercus tenuicolis* infects sheep in the same region

*Taenia olingojinei* Dinnik and Sachs, 1969
Host: Spotted hyena (one out of two cases)
Location: Intestine
Collection Station: 21–8. D.d. (Grabber and Coll, 1973 b)

*Taenia regis* Baer 1923
Host: Lion (Three out of three)
Location: Intestine
The corresponding Cysticercis is a parasite of the serous membrane pericardium. Pleura, peritoneum, mesentery) the hepatic parenchyma and th heart. It is found throughout the Cygegetic area (from the 5th to the 10th parallel) in wharthogs, water buck, cob de buffon, Reebuck, hippotragus, Damalis the bubal. The buffalo is not infected. Infestation rate: Wharthog 18%, Antelopes 9.9% (Troncy et al; 1972 a) Grabber et al; 1972 d)

*Taenia taeniæ formis* Batsch, 1786
Hosts: Black Rat, dorsal band balck rat.

Location: Intestine
Collection Station: Throughout the Centrafric Republic (Quentin, 1964c).

*Cysticercus bovis* (Larva form of *Taenia saginata in man*)
Host: Zebu, excluding wild ruminants
Location: Intermuscular joint
Collection Station: Throughout CAR. infestation rate for 15 to 30 month old calves is 21% and that of full grown bovine is 49% (Grabber 1961; Grabber and Thome, 1964; Farcha yearly report, 1968 Graber et al., 1969; Graber and Troncy: 1972.
Lion adapted *Echino Coccus granulosus granulosus* stock
Location: Lion
Hydatid cysts corresponding to this stock have been discovered in eastern CAR from the 5th to the 9th parallel, in the wharthog, the bushpig, excluding the buffalo and antelopes. Location: Liver, lung and heart. Infestation rate for wharthogs: 38% (Troncy and Coll. 1972 a) Graber and Thal, 1980). For the time being no hydatid cyst has been found in the liver and lung of Zebus, sheep and goats of C.A.R. except in Bangui, in a Zebus of Chadian origin (Grabber, 1961)

3.—) Nematoda
3–1—) Enoploida

*Trichuridae* Family Gedoelst, 1916

*Trichuris Carlirieri* Gedoelst, 1916
Host: Jackson rat
Location: Intestine
Collection Station: La Maboka (Quentin, 1966b)

*Trichuris globulosa* von linstow, 1901
Hosts and infestation rates: Zebus (juvenile 24%; adults 1.2%)
Location: Caeicum
Collection Station: Bouar (Grabber et al, 1969)

*Trichuris muris* Schrank, 1788
Hosts: Jackson rat; Mastomys SP; Eminrat
Location: large intestine
Collection Station: La Maboke (Quentin, 1966b)

*Capillaria baylisi* Quentin 1966
Host: bristled rat
Location: Intestine
Collection Station: La Maboke (Quentin, 1966b)
**Capillaria obsignata** Madsen, 1945  
Host and infestation rates: Chicken (24%)  
Location: Intestine  
Collection Station: Bangui and Bambari (Far-cha report, 1968)

**Capillaria pearsi** Baylis, 1926  
Host: Mstomys SP  
Location: Intestine  
Collection Stations: La Makobe (Quentin, 1966 b)

3–2—(Rhabditida)  
**Strongyloidae** Family chitwood and pclntosh, 1934

**Strongyloides avium** Cram, 1929  
Hosts and infestation rate: Chicken (40%)  
Location: Small intestine  
Collection Station: Bangui (Farcha Report, 1968)

**Strongyloides papillosus** Wedl 1856  
Host: Dairy Cow  
Location: duodenum  
Collection Station: Bouar (Bouchet et al, 1969 a and b)

**Parastrongyloides chrysochloris** Quentin, 1969  
Hosts: golden mole; bristled rat  
Location: Intestine  
Collection Station: la Maboke (Quentin, 1969 c)

3–3–3-) **Strongyloida**  
3–3–3–1–)  
**Diaphanocephalidae** family Travassos, 1920  
**Kalicephalus colubri** orlepp, 1923  
Hosts: Naja melanoleuca; Boaedon olivaceus  
**Bothrocephalus atractaspis irregularis**  
Location: Intestine  
Collection Station: Boukoko (Schad, 1962, Ghadirian 1968)

**Kalicephalus paracolubri** Ghadirian, 1968  
Hosts: Naja melanoleuca; Boaedon olivaceus  
**Bothrocephalus lineatus; Atractaspis irregularis**  
Collection Station: Boukoko (Schad, 1962; Ghadirian 1968)

3–3–3–2 **Ancylostomatidae** family Nicoll, 1927  
**Buonostomum trigonocephalum** Rudolph, 1808  
Hosts and infestation rates: Sheep (52%) goat.  
Location: duodenum  
Collection Station: Bouar (Farcha report, 1968)

**Buonostomum phlebotomum** Raellet, 1900  
Hosts and infestation rates: Zebu (juvenile 50%); adults 3.4%)  
Location: duodenum  
Collection Stations: Bouar Carnot, Bambari Bangui (Bouchet et al, 1969; Graber et al; 1969)

**Gaigeria pachyscelis** Railliet and Henry, 1910  
Hosts: Goats, Buffalo (twice)  
Location: Duodenum  
Collection Station: Bouar; 23–6. c.d.

**Graimocephalus clathratus** Baird, 1868  
Host and infestation rate: Elephant (all animals autopsied)  
Location: Bile ducts  

3–3–3–3-) **Strongyloidae** Family Baird, 1853  
**Strongyulus equinus** Muller, 1780  
Host: ASS  
Location: Colon and Caecum  
Collection Station: Bouar.

**Delaondia vulgaris** Looss, 1900  
Host: ASS  
Location: Colon and Caecum  
Collection Station: Bouar

**Triodontophorous minor** Looss, 1900  
Host: ASS  
Location: Colon and Caecum  
Collection Station: Bouar

**Grobocephalus urosubulatus** Alessandrini 1909  
Hosts: Pig; Bishpig  
Location: Large intestine  
Collection Stations: Bangui (Graber, 1961); 25.5. C.c Troncy et al, 1972 a).

3–3–3–4–) **Cyathostomidae** Family Yamaguti, 1961  
**Trichonema anriculatum** looss, 1900  
Host: ASS  
Location: Colon and Caecum  
Collection Station: Bouar

**Khalilia Sameera** Khalil, 1922  
Host: Elephant (two out of four cases)  
Location: Large intestine  

**Murshidia (Murshidia) longicaudata**  
Neveu—Lemaire, 1928  
Host: Elephant (one out of four cases)  
Location: Large intestine  
Collection Station: 22.8. D.a; 25.5 D.d. (Boulot, 1975)
**Murshidia (Pteridopharynx) memphisia** Khalil, 1922  
Host: Elephant (one out of four cases)  
Location: Intestine  
Collection Station: 24–6. B.d.  

**Murshidia (Chabaudia) hamata** Daubney, 1923  
Hosts: Wharthog, Giant forest-hog (rare)  
Location: Large intestine  
Collection Station: 25.5. D.d. (Troncy et al., 1972 a)  

**Murshidia puginicandata** (Leiper, 1909) Yorke and Mapleton, 1926  
Hosts: Wharthog; Giant Forest-hog  
Location: Large intestine  
Collection Station: 25.5. D.d. (Troncy et al., 1972 a)  

**Bourgelatia pricei** (Schwartz, 1924) Troncy, Graber and Thal, 1972  
Hosts: Wharthog, Giant forest-hog  
Location: Large intestine  
Collection Station: Cynegetic area of the 5th to 8th parallel (Troncy et al. 1972 a)  

**Bourgelatia hylochoeri** van den Berghe 1943  
Host: Giant forest hog  
Location: Large Intestine  
Collection Station: 25.5. D.d. (Troncy et al., 1972 a)  

**Quilonia magna** Neveu-Lemaire, 1928  
Host: Elephant (two cases out of four cases)  
Location: Intestine  

**Daubneya centrafricanum** Troncy, Graber and Thal, 1972  

**Daubneya eurycephalum**, Goodey 1924  

**Daubneya mpwapwae** Dulhy 1947  
Daubneya Roubandi Daubney 1926  
Host: Wharthog, with the exception of *Droubaudi*. They are rare and less abundant parasites.  
Location: Large intestine  
Collection Stations: throughout the Cynegetic area of Eastern CAR (Troncy et al., 1972 a and 1973 c)  

**Daubneya farchai** Troncy, Graber and Thal, 1972  

**Daubneya goodeyi** Daubney, 1926  

**Daubneya muanzae** Daubney, 1924  

**Daubneya yorkei** Thornton, 1924  
Hosts: Wharthog; Giant forest-hog. They are common and always largely representative Nematoda (except D Farchai)  

**Location:** Large intestine  
**Collection Station:** Cynegetic area of eastern CAR (Troncy et al., 1972a and 1973c)  

**Daubneya enzebyi** Troncy, Graber and Thal, 1972  
Host: Giant forest-hog  
Location: Large intestine  
Collection Station: 25.5. D.d. (Troncy et al., 1973 c)  

**Oesophagostomum (Oesophagostomum) dentatum** Rudolphli, 1803  
Host: Domestic pig  
Location: Large Intestine  
Collection Station: Bouar (Graber, 1961; Farcha report, 1968)  

**Oesophagostomum (proteracrum) colombianum** Curtice, 1890.  
Hosts and infestation rates: Sheep (60%) goat.  
Location: Colon and Caecum  
Collection Station: Bouar (Graber, 1961; Farcha report, 1968).  

**Oesophagostomum (proteracrum) Synceri** Troncy and Thal, 1977  
Host: Buffalo (three out of 74 cases)  
Location: Large Intestine  

**Oesophagostomum (Bosiocola) radiatum** Rudolphli, 1803  
Hosts and infestation rates: Zebu (juveniles 50% adults 40%)  
Location: Caecum and large intestine  
Collection stations: Bouar, Carnot, Bangui Bambari (Graber, 1968; Bouchet et al., 1969; Graber et al, 1969)  

3–3–5—*Stephanuridae* Family Travassos and Vogelsang, 1933 *Stephanurus dentatus* Diesing, 1839  
Hosts: domestic pig; wharthog (twice out of 74)  
Location: Liver, perirenal adipose tissue  
Collection Station: Bouar, Bangui (Graber, 1961); 23.6. C.c.; 21.8. D.d. (Graber, Euzeybi and Thal, 1971; Troncy et al, 1972 a)  

*Mammomonogamus nasica* von Linstow 1899.  
Hosts and infestation rates: Zebu (34%) vocal cord and trachea  
Collection Stations: Bouar (Vercruysse, 1978); 23.6 C.d. (Graber et al., 1971 and 1972a)
3–3–7–) Malaineidae Family (Skrjabin and Schultz; 1937) Durette — Desset and Chabaud, 1977

Allintschius dunni Durette — Desset and Chabaud, 1975
Host: Pygmy pipistrelle
Location: Intestine
Collection Station: La Maboke

Molineus teocchii Quentin, 1969
Host: Demidov's Galago
Location: Intestine
Collection Station: La Maboke (Quentin, 1969)

Molineus vogelianus de Muro 1933
Host: Potto
Location: Intestine
Collection Station: La Maboke (Quentin, 1965 a)

Molineus congoensis Adam and wanson 1954
Host: tree-dwelling rat
Location: Liver

Dollfusstrongylus sciuereis Quetin, 1970
Host: Reddish brown legged funisciure
Location: Intestine
Collection Station: La Maboke

Trichochoenia conincki Chaband, Bainer Puyllaert, 1967
Host: Tricuspid Scaled Pangolin
Location: duodenum
Collection station: Bamango (Prod'hon, 1969)

Trichochoenia rousseti Biocca, 1959
Host: Tricuspid Scaled pangolin
Location: Intestine
Collection Station: Bomango (Prod'hon 1969)

3–3–8–) Heligmosomidae Family (Travassos) (1914) Cram 1927

Longistriata mabokensis Durette-Desset, 1970

Longistriata albaretii Durette-Desset, 1970
Host; Reddish brown legged funisciure
Location: Intestine
Collection Station: La Maboke (Durette-Desset, 1970 a)

Longistriata paratrifurcata Durette-Desset, 1970

Longistriata quatanuda Durette-Desset, 1970

Longistriata posterior Durette-Desset, 1970

Longistriata rata Durette-Desset, 1970
Host: Striped funisciure
Location: Intestine
Collection Station: Maboke (Durette Desset, 1970 a and b)

Longistriata thamnomyssis Durette-Desset, 1966
Host: Emin rat; tree-dwelling rat
Location: duodenum
Collection Station: M'baiki; La Maboke, Boukoko (Durette-Desset, 1966)

Longistriata hylomys Durette-Desset, 1966
Host: Dorsal band black rat
Location: Intestine
Collection Station: Boukoko (Durette-Desset, 1966)

Longistriata petteri Durette-Desset, 1964
Hosts: Dorsal band black rat; Jackson rat, tree dwelling rat
Location: duodenum
Collection Station: Haute Sangha; Boukoko (Durette-Desset 1964 and 1966)

Longistriata chippauxi Durette-Desset, 1964
Host: Red muzzled rat of Gabon
Location: Intestine
Collection Station: Boukoko (Durette-Desset, 1964)

Longistriati chabaudi Durette-Desset, 1964
Host: Mastomys SP
Location: Intestine
Collection Station: Boukoko (Durette-Desset, 1964)

Longistriata dupuisi Durette-Desset, 1964

Longistriata heimi Durette-Desset, 1964
Host: Jackson rat
Location: duodenum
Collection Station: Boukoko (Durette-Desset, 1964)

Longistriata lemniscomysi Durette-Desset, 1970
Host: striped rat
Location: duodenum
Collection Station: La Maboke (Durette-Desset, 1970 d)

Longistriata petri Durette-Desset 1970
Host: Bush mouse
Location: duodenum
Collection Station: La Maboke (Durette-Desset, 1970 d)

3–3–9–) Heligmonelliidae Family (Skrjabin and Shikhobalova, 1952) Durette-Desset and Chabaud, 1977

*Heligmonella streptocerca* Baylis 1928
Host: Striped funiscure
Location: Intestine
Collection Station: La Maboke (Durette-Desset, 1970 a)

*Quentinstrongylus graphiüri* Durette-Desset, 1969.
Host: Silvery lerot
Location: Intestine
Collection Station: La Maboke

*Tenorastrongylus parvulus* Durette-Desset, 1966
Host: Mus Minutoides; *Mustriton*
Location: duodenum

*Cooperia punctata* von Linstow, 1907
Host: Zebu
Location: Small Intestine

*Cooperia pectinata* Ransom 1907
Host: Zebu
Location: Small Intestine
Collection Station: Bouar, Carnot, Bangui C. punctata and C. pectinata infect 52% of juvenile bovine and less than 10% of adults (Graber and Coll, 1969)

*Ostertagia thalae* Troncy and Graber, 1973
Hosts: Bubal, Hippotragus
Location: Petrel

*Haemonchus contortus* Rudolph, 1803
Hosts and infestation rates: Zebu (between 35% and 50%) Sheep (35%) Goat and Buffalo (one out of 74 cases)
Location: Petrel

*Ashworthius lerouxi* Diadure, 1964
Host and infestation rate: Buffalo (70%)
Location: Petrel

Collection Station: The entire cygenetic zone of eastern CAR.

3–3–11–) *Metastrongylidae* Family leiper 1908

*Metastrongylus salmi* Gedoelst, 1923
Host: Domestic pig
Location: Bronchi
Collection Station: Bouar (Graber, 1961)

3–3–12–) *Protostrongylidae* Family leiper 1926 *pneumostrongylus cornigerus* ortlepp 1962
Hosts: Hippotragus (4 out of 7) Damalisc; Bubal (6 out of 14)
Location: Pulmonary alveolus
Collection Stations: The 7th and 9th parallel cygenetic zone (Graber et al, 1973 c)


*Stefanskostrongylus pottoi* Chaband and Bain, 1965
Host: Potto
Location: Various viscerae, liver
Collection Station: Maboke (Chab and Bain, 1965; Chaband, 1972.

3–4–) *Oxyurida*

3–4–1–) *Oxyuridae* Family Cobbold, 1964

*Oxyurus equi schrank*, 1788
Host: ASS
Locations: Colon and Caecum
Collection Station: Bouar.

*Zenkoxyurus mabokensis* Quentin, 1974
Host: Apterous Anomalure
Location: Caecum
Collection Station: La Maboke (Quentin, 1974)

*Syphacia lophyromyos* Quetin, 1966
Host: Bristled rat
Location: Intestine
Collection Station: La Maboke (Quentin, 1971a)

*Syphacia nigeriana* Baylis, 1928
Hosts: striped rat; mastomys SP; Jackson rat; Bush mouse, water rat.
Location: Caecum
Collection Station: Maboke (Quentin, 1971a)

*Syphacia obveolata* Rudolph 1902
Hosts: Water rat; Jackson rat
Locations: Caecum and Rectum
Collection Station: Bangui (Quentin, 1966a)

3–4–2–) *Heteroxyenematidae* Family skrjabin and shikhobalova, 1948
Aspicularis tetraptera Nitzsch, 1821
Hosts: Mastomys SP; Jackson rat; tree-dwelling rat.
Locations: Caecum
Collection Station: Bangui, La Maboke (Quentin, 1966a)

Aspicularis Africana Quentin 1966
Hosts: Mastomys SP; Tree-dwelling rat
Location: Caecum
Collection Station: Bebe (Quentin, 1966a)

3–5–) Ascaridia
3–5–1–) Cosmocercidae family Railliet, 1916
Travassos, 1925 Raillietnema bainiae Petter, 1966
Host: Xerixys erosa
Location: Colon
Collection Station: La Maboke

3–5–2–) Atractidae Family (Railliet, 1917)
Travassos, 1919.

Probstmayria suis Tvoncy, Graber and Thal, 1972
Hosts: Whart-hog, Giant Forest hog.
Location: Large intestine
Collection Station: Throughout the cynecetic zone of CAR Troncy et al, 1972 a and b)

3–5–3–) Seuratidae family Hall, 1916
Schneiderinina chabaudi Quentin, 1965
Host: Bristled rat
Collection Station: Boukoko (Quentin, 1965c)

3–5–4–) Ascaridae Family Baird, 1853

Toxocara vitulorum Goeze, 1782
Host: Zebu, more than 50% of dairy veal of one to three months.
Location: Intestine
Collection Station: Throughout the animal husbandry zone of CAR (Graber, 1961 and 1968; Bouchet et al. 1969)

Toxocara canis werner, 1782
Host: Dog
Location: Intestine
Collection Station: Bouar

Parascarid equorum Goeze, 1782
Host: ASS
Location: Intestine
Collection Station: Bouar

Ascaris suum Goeze, 1782
Host: Domestic pig
Location: Intestine
Collection Station: Bouar (Farcha report, 1968)

Ascaris phacochoeri Gedoelst, 1916
Host and infestation rate: Whart-hog (one third of infested animals)
Location: small intestine
Collection Station: Throughout the cynecetic zone of CAR (Troncy and Coll, 1972 a)

Toxascaris vesterae warren, 1971
Host: Spotted hyena
Location: Intestine

Toxascaris leonina von linstow, 1902
Host: Lion
Location: Intestine

3–5–5–) Heterakidae Family Railliet and Henry, 1912.

Heterakis brevispiculum Gendre, 1911.
Host and infestation rate: chicken (10%)
Location: Intestinal Caecums
Collection Stations: Bouar; Bangui (Farcha report, 1968)

3–5–6–) Ascaridiidae Family Travassos, 1919

Ascaridia galli schrank 1788
Host and infestation rate: Chicken 39%
Location: Intestine
Collection Stations: Bouar, Bangui, Bambari
(Farcha yearly report, 1968)

Ascaridia numidae Leiper, 1908
Host: Guinea Fowl (39%)
Location: Intestine
Collection Station: Bouar.

3–5–7–) Subuluridae Family (Travassos 1919

Ascaridia galli schrank 1788
Host and infestation rate: chicken (39%)
Location: Intestine
Collection Stations: Bouar, Bangui, Bambari
(Farcha yearly report, 1968)

Ascaridia numidae Leiper, 1908
Host: Guinea Fowl (39%)
Location: Intestine
Collection Station: Bouar.

3.5–7–) Subuluridae family (Travassos 1914)
Yorke and Mapelstone, 1926.

Subulura otoolicii van Beneden, 1890
Hosts: Demidov's Galago; Allen's Galago
Location: Caecum
Collection Station: La Maboke (Quentin and Tcheprakoff, 1969)
**Subulura williaminglisi** Quentin, 1965
*Hosts:* Emin's Rat, Dorsal band black rat, tree, dwelling rat.
*Location:* Caecum
*Collection Station:* M'baiki, Boukoko (Quentin, 1965 c and 1969 d)

**Allopora suctoria** Molin, 1860
*Host:* Chicken
*Location:* Intestinal Caecums
*Collection Station:* Bouar

3–6–)

**Spirurida**

3–6–1) *Gnathostomatidae* family Railliet, 1895
*Gnathostoma hispidum* Fedtschenko, 1872
*Hosts:* Domestic pig; whart-hog
*Location:* Stomach
*Collection Stations:* Bangui (Graber, 1961); 25.5. C.C. (Troncy et al., 1972 a)

3–6–2–) *Physalopteridae* Family (Railliet 1893) Leiper, 1908

**Pseudophysaloptera vincenti** Quentin, 1969
*Host:* Demidov's Galago
*Location:* Stomach
*Collection Station:* Maboche (Quentin, 1969f)

**Pseudophysaloptera soricina** Baylis, 1934
*Host:* Crocidura SP
*Location:* duodenum
*Collection Station:* Maboche (Quentin, 1969f)

**Physaloptera praeputiale** von linstow, 1889
*Hosts:* Hyena, lion, panther. *Spiruridinae* present in all the animals examined.
*Location:* Stomach

**Physaloptera joyeuxi** Gendre, 1928
*Host and infestation rate:* whart hog 29%
*Location:* Stomach

3–6–3–) *Rictulariidae* Family (Hall, 1915)
Railliet, 1916

**Pteygodmatites dolfusii** Chaband and Rousselot, 1956
*Host:* Two-spotted Palm-civet.
*Location:* Small intestine
*Collection Station:* La Maboche(Quentin,1969h)

**Pteygodmatites desportesi** chaband and Rousselot, 1956
*Host:* Bristled rat
*Location:* Intestine
*Collection Station:* La Maboche (Quentin 1965b and 1969 b)

3–6–4–) *Congylonematidae* Family (Hall 1916) Soboldy, 1949

**Congylonema dupuisi** Quentin, 1965
*Host:* Mastomys SP
*Location:* Stomach
*Collection Station:* La Maboche (Quentin, 1965 b and 1969 b)

3–6–5–) *Spiruridae* family oerley, 1885

**Protospirura muricola** Gedoest, 1916
*Hosts:* Emin rat, Dorsal band black rat, Alexandria rat; Mastomys SP; Jackson rat;

**Praomys morio; Mus minutoides**
*Location:* Stomach
*Collection Stations:* M'baiki, Boukoko, La Maboche, Bebe, Toukoulou (Quentin, 1965 b and 1969 c).

3–6–6–) *Spirocercidae* family chitwood and wehr, 1932

**Spirocercus lupi** Rudolphi, 1809
*Host:* Dog
*Location:* Stomach
*Collection Station:* Bangui

**Cylicospirura** (*Clycospirura*) *subequalis* Molin, 1880) Vevers, 1922.
*Hosts:* spotted hyena; lion, panther. Present in all animals autopsied
*Location:* Stomach

**Ascarops stongyliina** Rudolphi, 1819
*Host Domestic pig
*Location:* Stomach
*Collection Station:* Bangui (Graber, 1961)

**Physoccephalus sexalatus** molin, 1860
*Hosts:* Domestic pig; whart-hog (one out of 74 cases)
*Location:* Stomach
*Collection Stations:* Bangui — (Graber, 1961); 21.8. D.d; 22.9. C.a. (Troncy et al., 1972 a)

**Physoccephalus SP**
*Host:* Black dorsal rat
*Location:* Stomach
*Collection Station:* Bouoko (Quentin, 1965 b)

3–6–7–) *Habronematidae* family

**Chitwood and wehr, 1932** Invaschkin, 1961.

**Habronema muscae** Carter, 1961
*Host:* ASS
*Location:* Stomach
*Collection Station:* Bouar
**Parabronema skrjabini** Rassovska 1924

**Hosts:** Hippotragus (two out of seven cases)

**Buffalo:** (one out of seven cases)

**Location:** Petrel

**Collection Stations:** 19.9. A.b; w21.8. D.d; 21.9. D.d. (Troncy and Coll; 1973 b)

*3–6–9–* Onchocercidae family Leiper, 1911

**Dirofilaria repens** Railliet and Henry, 1911

**Host:** Lion

**Location:** Sub-cutaneous joint

**Collection Stations:** 21.8.D.b (Graber et al, 1972 c)

**Loitomosapujoli** Bain, 1966

**Host:** wolly phylorrhino

**Location:** Abdominal cavity

**Collection Station:** La Maboke (Bain, 1966)

**Elaeophora poeli** vryburg, 1897

**Host and infestation rate:** Buffalo (13%)

**Locations:** Anterior and Posterior aorta.

**Collection Stations:** Throughout the 6th and the 9th parallel cynogenetic area (Graber et al, 1972 b)

**Elaeophora sagittata** von Linstow, 1907

**Hosts:** Buffalo (one out of seven cases) Derby Eland one out of three cases); bush-buck (two out of four cases)

**Locations:** Posterior aorta, heart, coronary veins.

**Collection Stations:** 23.7. A.d; 23.7. C.d; 23.7. D.c; 23.6. C.d. (Graber et al, 1972 b)

**Onchocerca armillata** Railliet and Henry, 1909

**Hosts:** Zebu; Buffalo

**Location:** Posterior aorta

**Collection Stations:** Bouar, Bangui (Graber, 1961; Graber et al, 1969) Cygenetic -zone of the 5th to 7th parallel (Graber et al, 1972 b)

**Onchocerca gutturosa** neumarn, 1910

**Host:** Zebu

**Location:** Cervical ligament

**Collection Stations:** Bouar, Bangui

**Setaria africana** Yeh, 1959

**Hosts:** Derby Eland; bush-buck

**Location:** Peritoneum

**Collection Stations:** 22.9. C.d.; 23.7. C.d.

**Setaria africana farchai** (Yeh, 1959)

**Troncy,** Graber and Thal. 1976

**Host:** Bush-buck

**Location:** Peritoneum

**Collection Stations:** 20.7. C.b; 20.6. C.d; 23.7. D.c.

**Setaria bicornata** von linstow, 1901

**Hosts:** Bohar Reedbuck; Common redbuck

**Location:** Peritoneum


**Setaria castroi** Ortlepp, 1964

**Hosts:** whart-hog (two cases out of 74 cases)

**Location:** Peritoneum

**Collection Stations:** 22.9. D.a; 22.9. C.a. (Troncy et al 1972 a)

**Setaria congolensis** Railliet and Henry, 1911

**Hosts:** Domestic pig, bush pig, giant forest-hog

**Location:** Peritoneum

**Collection Stations:** Bambari (Farcha report 1968) 20.7. D.a; 25.5. C.c.; 25.5. D.d (Troncy et al., 1972a)

**Setaria equina** Abildgaard, 1789

**Host:** ASS

**Location:** Peritoneum

**Collection Station:** Bouar

**Setaria graberi** shoho

**Host:** Reedbuck

**Location:** Peritoneum

**Collection Station:** 21.9. B.b. (Troncy, Graber and Thal, 1976).

**Setaria hornbyi brevicaudatus** Kreis 1938

Host and infestation level: Hippotragus (6 cases out of seven)

**Location:** Peritoneum


**Setaria labiatopapillos** Terroncito, 1882

**Hosts and infestation rates; Zebu (3%) Bubal;

**Location:** Peritoneum

**Collection Stations:** Bouar (Graber et al, 1969); 23.7. C.d. (Troncy, Graber and Thal, 1976)

**Setaria lamyfortensis** Troncy Graber and Thal, 1976

**Host:** Reedbuck

**Location:** Peritoneum

**Collection Stations:** 21.9. B.b; 21.10. B.b.

**Setaria nelsoni** shoho, 1976

**Host and infestation rate Buffalo (66%)

**Location:** Peritoneum

**Collection Stations:** Throughout the cynegetic zone of eastern CAR (Troncy, Graber and Thal, 1976) Auando and Bambari (Graber et al, 1964)

**Setaria pillersi** Twaiete, 1927

**Host:** Cob de Buffon
Location: Peritoneum  

*Setaria poultonti* Twai, 1927  
**Host:** Bubal (4 times out of 14)  
**Location:** Peritoneum  

*Setaria saegleri* levan Hoa, 1962  
**Host:** Crowned duiker  
**Location:** Peritoneum  
**Collection Stations:** 23.6. C.c.; 21.8.D.c. (Troncy, Graber and Thal, 1976)

*Setaria scaprum* Von Linstow, 1908  
**Host:** Oribi (4 times out of 5)  
**Location:** Peritoneum  
**Collection Stations:** 23.6. C.c.; 21.9. D.b. (Troncy, Graber and Thal, 1976) Birao and Poto-Poto (Graber et al. 1964)

*Dipetalonama dracunculoides* cobbold, 1870  
**Host:** Spotted hyena  
**Location:** Peritoneum  
**Collection Station:** 21.8. D.b.

4—) *Acanthocephalous*  
*Oligacanthorhynchidae* family South well and Macfie, 1925

*Ichadorhynchus quentini,* Troncy, 1969  
**Host:** spotted hyena  
**Location:** Intestine  

5—) *Poroccephalous*  
5—1— *Poroccephalidae* family heymons, 1922

*Armillifer armillatus* Wyman, 1847  
**Host:** Seba Python  
**Location:** Lung  
**Collection Station:** Bangui

*Armillifer armillatus* nymphs were collected from the serous membrane of the thoracic and abdominal cavities at the organ’s surface (heart, lung liver, spleen) or at the depth.

**Hosts and infestation rates:** whart-hog (15%) Bushpig, Giant forest-hog, Buffalo (3.1%) Civet; inchneumon mongoose lion, Panther and doguera baboon.

**Collection Stations:** The entire cyanetic zone of eastern CAR, from the 5th to the 9th parallel (Troncy et al, 1972a); Graber, Troncy and Thal, 1973 a)

*LINGUATULIDAE* family Heymons, 1935

*Neolinguatula nuttali* nymphs Sambon, 1922  
**Hosts and infestation rates:** Buffalo (30%) Cob defassa (1 out of 7 cases); Cob de buffon (1 out of 5) bush-buck (1 out of 4)

**Locations:** Circulatory System (Aorta, vein and hepatic artery, celiac trunk, right ventricle)  
Collection Stations. The entire cyanetic zone, of the 5th to the 8th parallel (Graber et al, 1972 b)

6 Agents of myiases  
6—1— *Oestridae family*  

*Kirkioestrus minutus* Rodhain and Bequaert, 1915  
**Host:** Bubal  
**Location:** Sinus and nasal cavities  
**Collection Stations:** Chad/CAR border (Graber and Gruvel, 1964; Graber and Thal, 1979 a)

*Kirkioestrus blanchardi* Gedoelst, 1914  
**Host:** Bubal  
**Location:** Sinus  
**Collection Station:** Lake mamoun, 21.10. B.b. (Graber and Thal 1979 a)

*Rhinoestrus phacochoeri* Rodhain and Bequaert, 1915.  
**Host and infestation rates:** whart-hog (25% to 45% according to the regions)  
**Location:** Sinus  
**Collection Stations.** The entire cyanetic zone of eastern CAR (Graber and Thal, 1979 a)

*Oestrus variolosus,* Loew, 1863  
**Host:** Damalisc  
**Location:** Sinus  
**Collection Station:** 21.8. D.a. (Graber and Thal, 1979 a)

*Oestrus ovis* linne 1761  
**Host:** Sheep  
**Location:** Sinus  
**Origin:** Bouar

*Gedoelstia cristata* Rodhain et Bequaett, 1913  
**Host:** Bubal  
**Location:** Sinus  
**Collection Stations:** Aouk and Lake Mamoun (Graber and Thal 1979 a).

*Strobiloestrus clarkii* clark, 1841  
**Host:** Reedbuck  
**Location Sub-Cutaneous back joint**  
**Collection Station:** Lake Mamoun (Graber and Thal, 1979 a)

*Gasterophilidae* Family  
Gasterophilus nasalis linne 1758
Host: ASS  
Location: Pylorus  
Collection Station: Bouar  

_Gasterophilus intestinalis_ De Geer, 1776  
Host: ASS  
Location: Stomach  
Collection Station: Bouar.

_Cobboldia loxodontis_ Brauer, 1897  
Host: Elephant (three cases out of four)  
Location: Stomach  
Collection Stations: Dinga (Graber and Gruvel, 1964); 25.5. C.d; 24.6. B.d. (Graber and Thal. 1979 a)

_Neocuterubra squamosa_ Grunberg, 1906  
Host: Elephant  
Location: Pad  
Collection Stations: East of CAR (Graber and Gruvel, 1964; Graber and Thal, 1979 a).

**LIST OF PARASITES AND THEIR HOSTS**

1—) **Carnivorous**

_CIVET, Civettictis civetta_  
_Armillifer armillatus_ Nymphs  

_TWO-SPOOTTED PALM-CIVET_ Nandinia bino-tata  
Pterygodermatites dolfusi

_ICHNEUMON MONGOOSE, Herpestes ichneu mon_  
Armillifer armillatus nympha

_SPOTTED HYENA, Haenea crocuta_  
_Taenia hyaenae_  
_Taenia crocutae_  
_Taenia olingoinei_  
_Toxascaris vesterae_  
_Physaloptera praeputiale_  
_Cyclicospirura (Cyclicospirura subequalis_  
_Dipetalonema dracunculooides_  
_Tchadorhyncus quentini_  

_LION, Panthera leo_  
_Diphyllobothrium theileri_  
_Joeyxieilla sp_  
_Taenia gonyamai_  
_Taenia regis_  
_Lion adapted Echinococcus granulosus granu-losus stock_  
_Toxascaris leonina_  
_Physaloptera praeputiale_  
_Cyclicospirura (Cyclicospirura) subequalis_  

_Dirofilaria repens_  
_Armillifer armillatus_ nymph  

DOG, _Canis familiaris_  
_Taenia hydatigena_  
_Toxocara canis_  
_SPIROCECA LUPUI_  

2—) **Artiodaetylous**

_DOMESTIC PIG, Sus Scrofa_  
_Gluborocephalus urosubulatus_  
_Oesophagostomum dentatum_  
_Stephanurus dentatus_  
_Metastrongylus salmi_  
_Ascaris suum_  
_Gnathostoma hispidum_  
_Ascarops strongylina_  
_Physcocephalus sexalutus_  
_Setaria congoensis_  

_WHART-HOG, Phaco choerus aethiopicus_  
_Fasciola giganteca_  
_Gastrudiscus aegyptiacus_  
_Choerocotyle epuluensis_  
_Moniezia mettami_  
_Seous Cysticercis of which the corresponding adult is Taenia regis of the lion_  
_Hydatid cyst whose corresponding adult could be a particular stock of lion adapted_  
_Echinococcus granulosus granulosus_  
_Murshidia (Chabaudia) hamata_  
_Murshidia (Chabandia) pugnicandata_  
_Bourgelatia pricei_  
_Daubneya centrafricanum_  
_Daubneya eurycepalum_  
_Daubneya farchai_  
_Daubneya goodeyi_  
_Daubneya mwanzae_  
_Daubneya mpwapwa_  
_Daubneya roubaudi_  
_Daubneya simpsoni_  
_Daubneya yorkei_  
_Stephanurus dentatus_  
_Probstmayria suis_  
_Ascaris phacochoeri_  
_Physaloptera joyeuxi_  
_Physcocephalus sexalutus_  
_Setaria castori_  
_Armillifer armillatus nympha_  
_Phinoestrus phacochoeri_  

_BUSHPIG, Potamochoerus porcus_
hydatid cyst whose corresponding adult could be a particular lion adapted
Echinococcus granulosus granulosus
Globocephalus urosubulatus
Gnathostoma hispidum
Armilla fer armillatus nymphs

GIANT FOREST HOG, *Hylochoerus meinertzhagei*
Choerocotyle epulensis
Moniezia mettami
Murshidia (Chabaudia) hamata
Murshidia (Chabaudia) pugnicandata
Bourgelatia pricei
Bourgelatia hylochoeri
Daubneyea enzebyi
Daubneyea enzebyi
Daubneyea farchai
Daubneyea goodeyi
Daubneyea mwanzae
Daubneyea yorkei
Probstmayria suis
Setaria congolensis
Armillifer armillatus nymphs

ZEBU, *Bos indicus*
Discococelium hospes
Fasciola gigantecia
Fasciola gigantecia
Schistosoma bovis
Paramphistomum microbothrium
Cotylophoron cotelphorum
Carmyrius spariosus
Carmyrius graberi
Carmyrius papillatus
Carmyrius parvapapillatus
Moniezia expansa
Moniezia benedeni
Thysaniezia ovilla
Echinococcus polymorphus (Bangui)
Cysticercus bovis
Trichuris globulosa
Strongyloides papillosus
Bonustomum phlebotomum
Oesophagostomum (Basciola) radiatum
Mammomonogamus nasicola
Cooperia pectinata
Cooperia punctata
Haemonchus contortus
Toxocara vitulorum
Parafilaria bovicina

Onchocerca armillata
Onchocerca gutturosa
Setaria labiatopapillosa

BUFFALO, bubalus, (Synterus) Caffer
Dicrocoelium hospes
Fasciola giganteca
Paramphistomum microbothrium
Paramphistomum clavula
Paramphistomum phillerouxi
Bothriophoron bothriophoron
Gigantocotyle symmeri
Cotylophoron macrophinctris
Carmyrius endopapillatus
Carmyrius exoporus
Carmyrius graberi
Carmyrius gregarius
Carmyrius papillatus
Carmyrius spariosus
Stephanopharynx coilos
Stephano pharynx compactus
Moniezia expansa
Avitelina centripunctata
Muscular cysticercis whose corresponding adult is *Taenia hyaenae* of hyenas
Gaigeria pachyscelis
Oesophagostomum (proteracrum) Synceri
Mammomonogamus nasicola
Haemonchus contortus
Ashworthius lerouxi
Elaeophora poeci
Elaeophora sagittata
Onchocerca armillata
Setaria nelsoni
Neolinguatula muttali nymphs
Armillifer armillatus nymphs

SHEEP, *Ovis aries*
Fasciola gigantica
Cotylophoron cotelphorum
Moniezia benedeni
Stilesia globipuneta
Avitelina centripunctata
Cysticercus tenuicollis
Bunostomum trigonocephalum
Oesophagostomum (proteracrum) Colembia
Haemonchus contortus

WATER BUCK, Cobus defassa
Fasciola gigantica
Paramphistomum microbothrium
Paramphistomum philleroxui
Cotylophoron Cotylophorum
Carmyrius papillatus
Carmyrius spatioiosus
Stephanopharynx Coilos
Stilesia hepatica
Setaria bicornata
Neolinguatula nuttali nympha

BUFFON COB Adonota Cob
Fasciola giganta
Parachistomum microbothrium
Parachistomum philleroxui
Cotylophorum
Carmyrius spatioiosus
Stephanopharynx Coilos
Avitellina Centripunctata
Stilesia hepatica

Mesentry and heart cisticercis whose corresponding adult is Taenia Regis of the lion.
Muscular cisticercis whose corresponding adult is Taenia hyaenae of the hyena Setaria pillersi neolinguata nuttali nympha

REEDBUCK, Redunca redunca nigeriensis
Paramphistomum clavula
Carmyrius graberi
Stephanoparynx Coilos
Mesentry cisticercis whose corresponding adult is Taenia regis of the lion
Setaria graberi
Setaria lamyfortensis
Strobiloestrus clarkii

ORIBI, Ourebia, ourebi
Paramphistomum microbothrium
Cotylophoron Cotylophorum
Cotylophoron microbothrium
Cotylophoron Cotylophorum
Cotylophoron macrospinchtris
Stilesia globipunctata
Setaria Scalprum

CROWNED COMMON DUCKER, Sylvicapra grimmia Avitellina
Centripunctata
Setaria Saegeri

HIPPRATUS, Hippotragus equinus
Faciola giganta
Paramphistomum microbothrium
Paramphistomum philleroxui

Gigantocotyle symmeri
Carmyrius exoporus
Carmyrius papillatus
Carmyrius spatioiosus
Stephanopharynx Coilos
Avitellina Sandgroundi
Stilesia hepatica

Muscular cisticercis whose corresponding adults are Taenia regis of the lion.
Mesentry, pericardium and pleura cisticercis whose corresponding adult is Taenia regis of the lion
Ostertagia thalae
Pneumostrongylus cornigerus
Parabronema Skjabinii
Setaria hornbyi brevicandatus

BUBAL, Alcelaphus lelwel
Fasciola giganta
Cotylophoron Cotylophorum
Cotylophoron macrospinchtris
Gigantocotyle symmeri
Carmyrius graberi
Carmyrius minitus
Carmyrius papillatus
Carmyrius spatioiosus
Avitellina Centripunctata

Muscular Cysticercis whose corresponding adult is Taenia hyaenae of the Hyena. Serous cysticercis whose corresponding adult is Taenia Regis of the lion.
Pneumostrongylus Cornigerus
Ostertagia Thalae
Setaria labiatopapillosa
Setaria poultoni
Kirkioestrus minitus
Gedoelstia Cristata
Damalisc, Damaliscus Korrigum
Cotylophoron Cotylophorum

Cysticercis of hepatic parenchyma whose corresponding adult is Taenia regis of lion
Pneumostrongylus cornigerus
Oestrus variolosus

BUSH BUCK: Tragelaphus Scriptus
Cotylophoron Cotylophorum
Carmyrius graberi
Carmyrius spatioiosus
Crossothrix baeri
Muscular cisticercis whose corresponding adult is Taenia Hyena of hyena
Elaeophora Sagittata
Setaria africana farchai
Neolinguatula nuttali nymphs

DERBY ELAND, Taurotragus derbianus
Carmynerus spathosus
Moniezia benedeni
Elaeophora sagittata
Setaria africana

3— Proboscidea
ELEPHANT, Loxodonta africana
Protoscoliola robusta
Brumptia bicaudata
Grammocephalus clathratus
Khalitia sameru
Quinonia magna
Murschidia (Murschidia) longicaudata
Murschidia (Pteridopharynx) Omoensis
Murschidia (Pteridopharynx) memphisia
Cobboldia loxodontis
Neocuterebra squamosa

4— Perissodactylyous
ASS, Equus asinus
Strongylus (Strongylus) equinus
Strongylus (Delafondia) Vulgaris
Trigondontoporus minor
TRichonema auriculatum
Oxyurus equi
Parascarisis equorum
Habronema muscae
Setaria equina
Gasterophilus nasalis
Gasterophilus intestinalis

5— Pholidota
TRICUSPID, SCALED PANGOLIN, Manis
tricuspid Trichochoenia Conincki
Trichochoenia rousseloti

6— Rodents
APTEROUS SPINY TAILED SQUIRREL, ZENKERELLA insignis
REDDISH BROWN LEGGED FUNICLE, Funisciurus phyrhopus
Dollfustrongylus Scurei
Longistriata mabokensis
Longistriata albaretii
REDDIS BROWN BACK STRIPED FUN- ICLE Funisciurus lemniscaius
Longistriata paratrifurcata
Longistriata quadranuda
Longistriata posterior
Longistriata rara
Heligmonella streptocerca
SILVERY LEROT, Graphiurus hueti Quentin
Strongylus
Congolencis
Trichuris muris
Longistriata thamnomysi
Subulura williaminglisi
protospirura muricola
NILE RAT, Arvicanthis niloticus
Inermicapsifer madagascariensis
BRISTLED BROWN RAT, Lophuromys Sikapusi
Skrjabinotaenia gerbilli
Anomotaenia heimi
Hymenolepis Petteri
Capillaria baylisi
Parastrongyloides Chrusochloris
Syphacia lophyromyos
Schneiderinema chabaudi
Pterigodermatides desportesii
STRIPED RAT, Lemniscomys striatus syphacia
nigeriana
Longistriata lemniscomysi
DORSAL BAND BLACK RAT, Hybomys
Univittatus
Skrjabinotaenia gerbilli
Inermicapsifer madagascariensis
Taenia taeniaformis
Molineus vogelianus
Longistriate hybomysi
Longistriata petteri
Subulura williaminglisi
Protospirura muricola
LONG TAILED WATER RAT, Stochomys
Longicaudatus
Skrjabinotaenia pauciproglossis
Syphacia nigeriana
Syphacia obveolata
RED MUZZLED RAT OF GABON, Oenomys
hypoxanthus
Inermicapsifer madagascariensis
Longistriata chippauxi
BLACK RAT, Rattus, rattus
Hymenolepis diminutu
Taenia taeniae formis
ALEXANDRIA RAT, Rattus Alexandrinus
Protospirura muricola
MASTOMYS SP
Catenotaenia lobata
Skrjabinotaenia lobata
Raiilietina baeri
Dilepis dolfini
Hymenolepis diminuta
Trichuris muris
Longistriata Chabaudi
Capillaria pearsi
Syphacia nigeriana
Aspicularis tetraperta
Aspicularis africana
Congylomena dupnisi
Protospirura muricola
JACKSON RAT, praomys jacksoni
Raiilietina baeri
Hymenolepis diminuta
Trichuris muris
Trichuris Carlieri
Trichuris petteri
Molineus vogelianus
Longistriata heimi
Longistriata petteri
Longistriata duplex
Syphacia nigeriana
Syphacia obvoleta
Aspicularis tetraperta
Protospirura muricola
PRAOMYS MORIO
SYPHACIA NIGERIANA
Protospirura muricola
MUS MINUTOIDES
Tenorastrongylius parvulus protospirura murica
MUS TRITON
Tenorastrongylius parvulus
HAILY TAILED TREE-DWELLING RAT
Thamnomys rutilus
Skrjabinotaenia baeri
Inermicapsifer madagascarensis
Molineus Congolensis
Molineus vogelianus
Longistriata Thamnomysi
Longistriata petteri
Aspicularis africana
Aspicularis tetraperta
Subulura williamlingisi
BUSH MOUSE, Hylomyscus Stella
Skrjabinotaenia media
Longistriata petri
Syphacia nigeriana
BATES RAT, Prionymys batesi
Skrjabinotaenia pauciproglottis
7—) Insectivorous
GOLDEN MOLE, Chrysochloris leucorhina
Parastrongyloides chrysochloris
Molineus adami
CROCIDURA SP
Pseudophaloperta soricina
8—) Chiroptera
WOLLY PHYLRHINE, Hipposideros Cyclops
Litomosoa pujili
PYGMY PIPISTRELLE, Pipistrellus nanus Allintoschius dunni
9—) Primates
DEMIDOV'S GALAGO, Galagoides, Demidovii
Molineus teocchii
Subulura otolinici
Pseudophaloperta vincenti
ALLEN GALAGO, Galago allenii
Subulura Otolinici
POTTO, Perocticus potto
Molineus vogelianus
Stefankostrongylius pottoi
DO GUERA BABOON; Papio anubis
Armillifer armillatus nymphs
MONKEY SP
Bertiella studeri
10—) Birds
FOWL: Gallus domesticus
Raiilietina (Raiilietina) Tetragona
Raiilietina (Raiilietina) echinobothrida
Hymenolepis Caroica
Choanotaenia infundibulum
Capillaria obsignata
Strongyloides avium
Ascaridia galli
Heterakis brevispirculum
Allopora Sactoria
GUINEA FOWL, *Numida meleagris*
Porogynia paranai
Cotugnia meleagris
\textit{Rallietina (Rallietina) pintneri}
Ascometra numida
Ascaridia numidae

LITTLE BEARDED GRIVELE, *Pongoiulius Scolopaceus*
Brachylaima attenuatum

YELLOW BEARDED BULDUL, *Andropodus latrorstris*
Brachylaima attenuatum

11— Amphibians

\textbf{CHIROMANTIS RUFESCENS}
Mesocoelium monas

\textbf{PTYCHADENA SUPERCILIARUS}
Mesocoelium gabonensis
Mesocoelium monas

\textbf{PTYCHADENA PERRETI}
Mesocoelium monas
Mesocoelium gabonensis
Mesocoelium monas

\textbf{PTYCHADENA OXYRHYNCHUS}
Polystoma prudhoei
Mesocoelium gabonensis
Mesocoelium monas

\textbf{HYLARANA ALBOLABRIS ALBOLABRIS}
Polystoma africanum gabonensis
Maederia aburnense
Mesocoelium monas

\textbf{AFRIXALAS FULVOVITTATUS}
POLYSTOMA UW
Polystoma Uwuwni

12— Reptiles

\textbf{KINIVYS EROSA}
Rallietinae bainae

\textbf{SEBA PYTHON, Python Sebae}
Armillifer Armillatus

\textbf{BOAEON OLIVACEUS}
Kalicephalus Colubri
Kalicephalus paracolubri

\textbf{BOTHROPTALMUS LINEATUS}
Kalicephalus Colubri
Kalicephalus paracolubri

\textbf{NAJA MELANOLEUCA}
Kalicephalus Colubri
Kalicephalus paracolubri

\textbf{ATRACTASPIS IRREGULARIS}
Kalicephalus colubri
Kalicephalus paracolubri

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Received for publication on 8th April, 1980
THE SUSCEPTIBILITY OF YOUNG SHEEP AND GOATS TO AN EXPERIMENTAL TRYPANOSOMA CONGOLENSE INFECTION

L. GRIFFIN* and E.W. ALLONBY,
Veterinary Services Division, P.O. Kabete.

SUMMARY

Young indigenous breeds of sheep and goats, varying in age from a few days to eight weeks, were experimentally infected with *Trypanosoma congoense*, and the course of disease and mortality rate compared with similarly infected adults. The clinical effects and mortality rate indicated that the young animals are more susceptible to the infection than the adults. The inclusion of young animals in a prophylactic regime as soon as they are grazed in endemic trypanosomiasis areas is suggested.

INTRODUCTION

The ability of calves to withstand infection with African trypanosomes more efficiently than adults under natural and experimental conditions has been reported by many authors. Fiennes, Jones and Laws (1946) compared the clinical pathology, haematology and morbid anatomy of *Trypanosoma congoense* infected calves with infected adults and reported that calves showed a blood picture more closely resembling that of uninfected controls than adults. Fiennes (1953) demonstrated that calves born of mothers protected by Antrycide remained refractory to infection for a longer period than untreated controls when exposed to natural challenge. Whiteside (1962) also reported that the calves of cows in an endemic area took longer to become infected than the longer the cows had been in the area.

Although Fiennes (1970) was convinced by these and other studies that young calves have greater powers to resist the disease than the adults, other workers (Wilson, Paris and Dar, 1975) report that significant resistance to trypanosomiasis did not develop in either calves or adults in a herd of breeding cattle maintained in an area of high challenge for over 3 years. Furthermore, the animals studied by Whiteside (1962) and Fiennes (1953) were on drug therapy so that the possibility of drug residues being passed to the young must be considered. Finally the available information on reduced susceptibility of young calves to trypanosomiasis is derived largely from field reports rather than from carefully controlled experiments (Terry, 1976).

We describe here work that was undertaken to investigate whether or not this phenomenon of reduced susceptibility of young animals to African trypanosomiasis also applies to sheep and goats experimentally infected with *Trypanosoma congoense*.

MATERIALS AND METHODS

Study area

The study was conducted at Kiboko Range Research Station, 160 km southeast of Nairobi in the Machakos District of Kenya, and at 1000 metres above sea level. The grazing area was composed mainly of open grass savannah and *Acacia-Commiphora* bushland, with a mean annual rainfall of 600 mm. The locality supported abundant game species grazing the same area as the domestic stock. Tsetse species in the area

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*Present address: P.O. Box 30020, Nairobi.
included *Glossina pallidipes*, which was the most common and widespread, *G. longipennis* in low density and *G. brevipalpis* is isolated localities.

**Experimental Animals**

Lambs and kids from the breeding flocks at Kiboko Range Research Station were used. The lambs were the first cross between Dorper rams and Blackhead Persian ewes, while the kids were pure-bred Galla. The dams had been on prophylactic therapy (isometamidium chloride) since their arrival at the station many months earlier. The young animals, varying in age between 1 day and 7 weeks, were retained in the pen with the dams for the first two or three days after birth. When the young were approximately two weeks old they were allowed to graze out in the bush with the flock and all the animals were penned together at night.

**Parasites**

A donor goat which had become naturally infected with *T. congolense* was used as the source for infection. When the parasitaemia was high, blood was taken from the jugular vein into EDTA and 0.5 ml inoculated immediately into the jugular vein of each experimental animal. The level of parasitaemia of the donor blood was measured using a haemocytometer.

The animals of each species were divided into six groups. Groups I to V were inoculated intravenously with $5 \times 10^5$ trypanosomes while Group VI was retained as uninfected controls.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>No. of sheep</th>
<th>No. of goats</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0–1 week</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>2–3 weeks</td>
<td>11</td>
<td>5</td>
</tr>
<tr>
<td>III</td>
<td>4–5 weeks</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>IV</td>
<td>6–7 weeks</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>V</td>
<td>Adults</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>VI</td>
<td>0–7 weeks</td>
<td>10</td>
<td>7</td>
</tr>
</tbody>
</table>

**Clinical Observations**

The course of the disease in each animal was monitored by weekly assays of the packed cell volume (PCV) and temperature readings, and the infection monitored by weekly Giemsa stained thick blood smear examinations.

**RESULTS**

**Packed Cell Volume**

From Table I showing the mean PCV of the animals during the course of the infection, it can be seen that in both the lambs and kids infected within the first week of birth the PCV decreased from between 44 and 43% to 29.5 and 29%, a decrease of 14.5% and 14.0% respectively.

Those animals infected at two or three weeks of age suffered a similar decline in PCV, 14.0% in lambs and 20.5% in kids. The older lambs and kids, particularly the latter showed a drop in PCV during the course of the infection of 17% in the lambs and more than 20% among the kids.

The infected adults showed the same PCV as the older lambs and kids by the end of the experiment, 17%, but the decrease during the course of the disease was only 11 and 12% among the sheep.
<table>
<thead>
<tr>
<th>Age at Infection</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
<th>Mean SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young Uninoculated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adults 6 to 7 weeks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adults 4 to 5 weeks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kids 2 to 3 weeks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kids 0 to 1 week</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Mean PCV of Lambs and Kids Given an Experimental Infection of T. congolense at various ages.
and goats respectively. The uninfected lambs and kids showed a drop in PCV of only 5% from 44 and 43% to 39 and 38% respectively. This decrease represents the normal change in PCV in uninfected growing animals.

**Temperature**

All the groups of infected animals showed an increase in temperature during the course of the disease. The mean temperature of each group at the start of the experiment was slightly above 101°F, but during the course of the infection increased by as much as 3.0°F in the young animals and 3.6°F in the adults, although the fluctuation in the temperature of individuals was even greater than the mean figures show.

**Infection Rate**

The rate of infection in each group reached 100% by the 4th week of the study. All infected animals including the adult controls showed a heavy parasitaemia by the 4th week after inoculation with *T. congolense*, but in most cases parasites were detected within two weeks.

**Mortality Rate**

There was a higher mortality rate among the youngest animals infected than among the older lambs and kids and adults. Six of the lambs and 3 of the kids infected at 1 to 3 weeks of age died during the 4 weeks of the study (Table 2). Of the older animals only 1 lamb infected at 4 to 5 weeks and one goat infected at 6-7 weeks died before the 5th week. The remaining animals were treated with diminazene aceturate because they were looking severely recumbent, and further loss from the breeding flock was undesirable. After treatment all animals made a good recovery. The control adults were still alive by the 5th weeks and improved after treatment.

**DISCUSSION**

Our results indicate that in the breeds of sheep and goats used, no greater resistance was exhibited by the young animals infected by needle passage with *T. congolense*. Indeed the very young animals suffered more severely from the disease as shown by the drop in PCV and the elevated temperature and showed a higher mortality rate than the older animals. Since the indigenous breeds of sheep and goats have been found to be more tolerant of experimental and natural *T. congolense* infection than exotic breeds (Griffin and Allonby 1979 a and b), it is possible that the young of exotic breeds are even more susceptible.

Most of the earlier reports of resistance among calves come from field studies where infection is with metacyclic forms in tsetse bites rather than by needle passage with trypomastigotes.

---

**Table 2:** The mortality rate in groups of young lambs and kids, five weeks after infection with *T. congolense*.

<table>
<thead>
<tr>
<th>Weeks after infection with T.c</th>
<th>Age at time of infection with <em>T. congolense</em></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 — 1 week</td>
</tr>
<tr>
<td>I Sheep</td>
<td>37.5% (3/8)</td>
</tr>
<tr>
<td>II Goats</td>
<td>33.3% (1/3)</td>
</tr>
</tbody>
</table>
Under field conditions it is possible that calves being smaller than adults are less frequently bitten by tsetse flies. Alternatively the immune response may act against the metacyclics at the time of infection limiting the establishment of parasites in the blood-stream.

Finally it is possible that in cattle drug residues passed in maternal milk may contribute to the resistance observed in calves (Urquhart and Murray, unpublished), although from our results this does not seem to apply to sheep and goats as all the ewes were on prophylactic therapy. However in view of the severe disease produced in the experimentally infected lambs and kids, as may occur during mechanical transmission it may be considered advisable to include young animals in a prophylactic regime as soon as they are exposed to tsetse challenge. Clearly comparisons between experimentally and naturally acquired infections in lambs and kids as well as in calves need to be made to determine the importance and mechanisms of the resistance or susceptibility of young animals to trypanosomiasis.

ACKNOWLEDGEMENTS
We are grateful to the staff of the Range Management Station and of the Tsetse Survey and Control Station at Kiboko for their technical support and daily co-operation, and would like to thank the Director of Veterinary Services Division for permission to publish the work. The UNDP/FAO Sheep and Goat Development Project provided the experimental animals and jointly financed the study.

REFERENCES

Received for publication on 7th March, 1980
A TSETSE SURVEY OF LUUKA AND KIGULU COUNTIES OF SOUTH BUSOGA DISTRICT, UGANDA, DURING AN OUTBREAK OF AFRICAN SLEEPING SICKNESS

S. B. KUTUZA* and JOSUE O. OKOTH,
East African Trypanosomiasis Research Organization, P.O. Box 96, Tororo, Uganda.

SUMMARY

A tsetse survey of Luuka and Kigulu countries of South Busonga, Uganda was carried out in October, 1976 to determine tsetse vectors involved in the transmission of trypanosomiasis. Only Glossina fuscipes fuscipes were found and none out of 191 caught, and divided into 16 batches, showed infection by feeding on albino rats. However, by trituration of the same batches one brucei complex trypanosome was isolated, giving a trypanosome infection rate of 0.52%, assuming that only one tsetse was infected in that batch. The results of the identification of blood meals revealed that G. f. fuscipes was feeding mainly on reptiles man and cattle also formed significant hosts.

INTRODUCTION

The area is of an undulating nature lying between 0° 37’ to 0° 45’N and 33° 23’ to 33° 27 E at an altitude between 1,000 and 1,200 metres above sea level. The area receives an average annual rainfall of 125 cm. The mean annual maximum and minimum temperatures are 27.5° and 15.0° C respectively. The area is drained by River Lumbuye and its tributaries which flow northwards into Lake Kyoga. Due to the agricultural practices of the people living in this area much of the natural forest has been destroyed and all that is left of it form narrow strips along the water courses. In some places the natural evergreen forest has been completely cleared and replaced by coffee, banana and sugarcane plantations. Man comes into contact with tsetse during animal hunting, cultivation, watering herds, charcoal burning, honey searching, crossing from one village to another, collecting water and firewood, and fishing (Fig. 2).

MATERIALS AND METHODS

Tsetse flies were caught from eight areas shown in Fig. 1 by random catches using hand nets. At the beginning, attempts were also made to catch tsetse off cattle when cattle were grazing in the vicinity of the study areas but no tsetse were seen alighting on them so the method was abandoned.

Hungry flies were sexed and put in Geigy-cages 15 x 10 x 5cm, fifteen flies per cage. Filled up cages were kept in a cool portable box. The gut contents of gorged flies in hunger stages one or two were expressed on Whatman filter paper No. 1 for the determination of their hosts. These blood smears were allowed to dry in air in the field and on returning to station were stored in a dessicator over calcium chloride. These were later sent to Dr. P.T.L. Boreham at the Imperial College Field Station, Ascorts, UK, for serological analysis of the hosts.

Tsetse were separated into batches according to sex and area. Twenty
Fig. 1. Map of Kigulu and Luuka counties of Busoga, Uganda showing area of survey

Fig. 2. Men in close contact with *Glossina fuscipes fuscipes* while fishing in river Lumbuye
was chosen as the maximum number per batch but where the numbers were too small, both sexes were combined (Table 1). Two techniques were employed to detect trypanosome infections in tsetse. Each batch of flies was allowed to feed on a clean albino rat for fifteen minutes. Counts of fed tsetse in each batch was made and where tsetse were reluctant to feed extra time and attention was given till all flies fed. Later the flies were anaesthetised with ether and triturated in borate buffer pH 8.0, one ml per fly. The supernate from each triturated batch of flies was inoculated into five white mice at 0.5 ml per mouse intraperitoneally. This was followed by an injection of cyclophosphamide at 50mg/kg of body weight immediately after challenge and thereafter daily for three days to suppress immune response of mice and rats. Mice received 0.25 ml of 4mg/ml solution in distilled water and rats received 1.0 ml of 10mg/ml solution also made up in distilled water. The rats weighed 200gm average and mice 20gm. The parasitaemic rats and mice were examined for the presence of trypanosomes for forty days after which they were discarded. Blood from parasitaemic animals was stained with Giemsa for the determination of the infecting species of trypanosomes. Where *brucei* subgroup trypanosomes were detected in the stained preparations the trypanosomes were cryopreserved in the liquid nitrogen. Only one isolate, designated EATRO 2306 afterwards, was found. This was tested by the BIIT (Rickman and Robson, 1970).

Two standards EATRO 2240, a derivative of proven *T. rhodesiense*, and EATRO 2238, *T. brucei sense stricto* were used together with buffer control. The isolate behaved like *T. rhodesiense*.

RESULTS

*Glossina fuscipes fuscipes* was the only tsetse caught. A summary of the isolation of trypanosomes is given in Table 1. Out of 191 *G.f. fuscipes* triturated in 16 batches, only one *T. brucei* complex was isolated giving an overall trypanosome infection rate of 0.52%. No trypanosomes were isolated by the rat-feeding method.

The results of the serological analysis of the blood meals (Table 2) indicated that reptiles provided most of the feeds (54.5%). Ox and man were also fed upon at 27.3% and 18.2% respectively.

### Table 1: Isolation of trypanosomes in tsetse from Luuka and Kigulu counties, south Busoga, Uganda.

<table>
<thead>
<tr>
<th>County</th>
<th>Location</th>
<th>Tsetse</th>
<th>T. brucei infection</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>Kigulu</td>
<td>Lugobango</td>
<td>11</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Naluko</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Nawankwale</td>
<td>38</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Nsinside</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Walugongo</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>Luuka</td>
<td>Bukade</td>
<td>24</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>Bukwanga</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>Lwaki</td>
<td>6</td>
<td>8</td>
</tr>
</tbody>
</table>
Table 2: Hosts of G. F. Fuscipes in Luuka and Kigulu counties, south Busoga, Uganda.

<table>
<thead>
<tr>
<th>Hosts</th>
<th>Bukade</th>
<th>Nsinze</th>
<th>Naluko</th>
<th>Nawankwale</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Man</td>
<td>—</td>
<td>9.1</td>
<td>9.1</td>
<td>—</td>
<td>18.2</td>
</tr>
<tr>
<td>Ox</td>
<td>9.1</td>
<td>9.1</td>
<td>—</td>
<td>9.1</td>
<td>27.3</td>
</tr>
<tr>
<td>Reptile</td>
<td>27.25</td>
<td>—</td>
<td>18.15</td>
<td>9.1</td>
<td>54.5</td>
</tr>
</tbody>
</table>

Only areas from where gorged flies were obtained are shown.

DISCUSSION

Glossina fuscipes fuscipes is probably one of the vectors involved in the transmission. The trypanosome infection rate of 0.52% was recorded. Rogers (1972) working in Bugweri county 15 kilometres east of the survey area found 4.8% infection rate of T. brucei subgroup organisms in G.f.fuscipes by trituration and mice feeding. Taylor (1930) working in Northern Nigeria recorded 0.14% infection rate in G. palpalis by dissection. A comparable infection rate (0.31%) was also obtained by Onyango et al (1964) when working during an outbreak in Central Nyanza, Kenya. He used trituration method.

In this survey there is a possibility that the infection rate was higher than actually recorded. The sample size used here was too small. The low infection rate in the tsetse might also have been due to the sporadic nature of the outbreak.

Glossina fuscipes fuscipes in this area was depending more on reptiles though they readily also fed on man and his domestic animals.

In the area under discussion man's occupation brings him into close contact with G. f. fuscipes. The popular Lantana hedge possibly provided shade for the tsetse. Onyango et al. (1966) found Glossina fuscipes fuscipes living in shades provided by Lantana shrubs. Chorley (1944) found the same Glossina breeding 12 miles away from water or lake. This suggests the extent to which this tsetse species can spread and increase the risk of trypanosome transmission to man.

ACKNOWLEDGEMENTS

We thank the Director, East African Trypanosomiasis Research Organization, for permission to publish these findings. We are also indebted to Miss E.A. Opiyo for carrying out the BIIT test and Dr. P.E.L. Boreham of the Imperial College Field Station, Ascorts, UK for serological analysis of the hosts.

REFERENCES


Received for publication on 6th October, 1978
DETECTION OF A TOGA VIRUS-LIKE AGENT IN BOVINE LEUKOCYTES.

I.E. HAJER*, A.W.A. MAHMOUD and O.A. ALI,
Central Veterinary Research Laboratory, Soba, Khartoum, Sudan.

SUMMARY

In the course of investigations to determine the association of rinderpest virus with circulating bovine leukocytes during viremia, virus-like particles were visualized in leukocytes obtained from a one year old calf. This calf, reportedly, was naturally infected with rinderpest. The virus-like particles were observed in the cytoplasmic matrix and within vacuoles of heavily infected leukocytes. Particles about 40 nm in diameter were observed within membrane-bound cytoplasmic structures (viral factories) containing an amorphous electron dense material (viroplasm). Larger 55-65nm spherical particles were visualized in the cytoplasmic matrix and vacuoles. Some of these particles seemed to be enveloped. The presence of different particle forms in different cytoplasmic sites probably reflected different stages of viral morphogenesis. The morphology and the morphogenesis of this agent is comparable to either group B arbovirus or pestivirus genus of Togaviridae. This viral agent is believed to be bovine virus diarrhoea virus (BVDV) because of its infectivity to cattle, its indistinguishable clinical symptoms from rinderpest and its affinity to bovine leukocytes.

INTRODUCTION

The differential diagnosis of rinderpest and rinderpest-like diseases such as BVD, bluetongue, malignant catarrhal fever and infectious bovine rhinotrace is important (Flowright 1968).

It has been established that both BVD and rinderpest viruses have particular affinity for lymphocytic tissues. Although it is usual practice to isolate rinderpest virus from theuffy coat of infected blood, the association between the virus and the circulating leukocytes has not been studied electron microscopically. Likewise, the association of BVDV with circulating leukocytes is not known.

The formation of viral nucleocapsid cores in the cytoplasmic matrix or vacuoles and their maturation by budding into cytoplasmic membranes have been described with Togavirus infecting insect, chick embryo and mammalian cells (Morgan et al, 1961; Acheson and Tamm, 1967; Whitefield et al., 1971). In this report, an electron microscopic evidence, based on morphogenesis, shows that a case clinically considered to be rinderpest was found to be Togavirus infection.

MATERIAL AND METHODS

A one-year calf (primary calf) was brought to the laboratory for autopsy and sample collection. The calf was selected as a representative case from a rinderpest outbreak.

Ten millilitres of blood were collected from this calf, 4 days after onset of the disease, in 10 ml of 0.01M ethylenediamine tetraacetic acid (EDTA). The blood was immediately centrifuged at 1,500 r.p.m. for 15 minutes. The buffy coat was collected and washed three
times by resuspension in phosphate buffered saline (PBS) and re-centrifugation at 1,500 r.p.m. for 15 minutes each time. The washed leukocytes were fixed in 2% glutaraldehyde for 2 hours and in 1% OsO₄ for 1 hour. Fixed specimens were dehydrated in an ascending graded series of ethanol and were impregnated with propylene oxide. The embedding medium was Epon 812. Thin sections were cut with glass knives using a LKB ultratome. The sections were stained with uranyl acetate and lead citrate (Reynolds, 1963), and examined with a Universal electron microscope E100-B, operated at 75 KV.

10 ml of a 10% spleen suspension from the primary calf were inoculated intravenously into two rinderpest hyperimmunized calves. The two calves were observed for clinical signs over a period of three weeks. Blood was collected when they reacted with fever. This blood was processed for electron microscopy as described above.

Primary bovine kidney cells were inoculated withuffy coat preparations from the primary calf. Three blind passages were carried out in primary bovine kidney cells using both tissue culture fluid and ruptured cells as inocula. The inoculated tissue cultures were observed for signs of cytopathic effect (CPE) for a period of 10 days each time.

Inoculation. These symptoms lasted for 3-4 days and both calves eventually recovered.

Attempts to isolate the viral agent in tissue cultures were unsuccessful.

The most common features of infected leukocytes from the primary calf were degeneration and lysis (Fig.1). None of the sections examined showed any evidence of rinderpest virus infection. Instead particles about 40 nm in diameter were observed embedded in, or in juxtaposition with an amorphous, electron-dense material (viroplasm) within membrane-bound cytoplasmic structures (viral factories) Fig. 2 & 3). Many distinctly larger, 55-65 nm, spherical particles were visualized in the cytoplasmic matrix and within membrane-bound cytoplasmic vacuoles. Some of the particles within these vacuoles seemed to be enveloped (Fig. 3 & 4). However, no cell membrane changes indicative of viral budding process were observed. In contrast, examination of leukocytes collected from the 2 rinderpest immune calves, either before or after inoculation, did not reveal any viral particles.

**DISCUSSION**

The symptoms exhibited by the primary calf could be considered typical of rinderpest infection; but other rinderpest-like diseases could not be excluded. Since rinderpest hyperimmunized calves reacted to inoculation with this agent, and since all sections from the primary calf did not show any evidence of rinderpest virus infection, and each section contained variable amounts of the particles described above, we believe that this viral agent was the primary pathogen in this case.

The particles observed were believed to be virions because their shape, size
Fig. 1. Infected leukocytes from the primary calf containing numerous viral particles (VP) and showing degeneration and lysis. (PL) = Plasmalemma; Bar = 1μ; Magnification X 10,500.

Fig. 2. Infected leukocyte from the primary calf. Viral factory containing numerous 40 nm particles (VP) in juxtaposition with an amorphous, electron dense viroplasm (VPL). Plasmalemma (PL) is intact. Bar = 1μ; Magnification X22,000.
Fig. 3. Infected leukocyte from the primary calf. 40 nm particles (VP) embedded in membrane-bound viral factory. 55-65 nm spherical viral particles within cytoplasmic vacuoles (CV). Some of the viral particles seem to be enveloped (EVP). Bar = 1μ; Magnification X20,000.

Fig. 4. Inset of rectangle in Figure 3. Detailed structure of viral Cores (VC). Some particles within cytoplasmic vacuoles seem to be enveloped (EVP). Bar = 0.5μ; Magnification X40,000.
and intracytoplasmic distribution distinguished them from any other cell organelles found in normal leukocytes. Although it is difficult to draw any dynamic conclusions from such an in vivo system, our observations indicated a probable sequence of viral replication events. The 40 nm particles were probably nucleocapsid cores formed from the electron dense material within viral factories. These cores matured by budding through cytoplasmic vacuoles to form the 55-65 nm particles. Similar viral replication patterns have been described for Togaviridae infecting insect, chicken embryo and mammalian cells (morgan et al., 1961; Acheson and Tamm, 1967; Whitefield et al., 1971). Paracrystalline arrays and lining of vacuoles with viral particles as observed for group A arboviruses (Bergold & Weible, 1962; Filshie & Rehacek, 1968; Whitefied et al., 1971) were not observed. The size, shape (Bergold & Weible, 1962) and the pattern of replication would identify this agent as either group B arbovirus or pestivirus genus of Toga-

viridae (Andrews et al., 1978).

It has been established that both rinderpest virus and BVDV have particular affinity to lymphocytic tissues and cause severe necrosis of leukocytes (Peter et al., 1968; Plowright, 1968). BVDV infection in cattle may be clinically indistinguishable from rinderpest because they both involve the digestive and respiratory tracts and cause diarrhoea, oronasal discharge, lacrimation and respiratory distress. Since BVDV is the only known Togavirus that infects cattle (Andrews et al., 1978) we believe that the viral agent reported herein is in all probability BVD virus.

REFERENCES


Received for publication on 8th January, 1980
EXPERIMENTS WITH A COMBINED VACCINE FOR POULTRY

D.R. NAWATHE and S.O. AYOOLA,
National Veterinary Research Institute, Vom, Nigeria.

SUMMARY

The LaSota strain of Newcastle disease virus, the Beaudette strain of fowl pox virus and the Weybridge strain of fowl typhoid were combined in a single vaccine for intramuscular administration to birds. The vaccine was safe and stable. Using birds of different ages the best antibody response to Newcastle disease virus was obtained by vaccinating at 4 and 8 weeks of age. Agglutinating antibodies of fowl typhoid were demonstrable as early as 10 days post vaccination, but the immunity to fowl pox virus was only assessed by challenge. Birds withstood challenge by Newcastle disease virus for up to 73 weeks following double vaccination.

INTRODUCTION

Multivalent, mixed or combined vaccines for poultry have been described by many workers e.g. Gupta and Rao (1969), Provost and Borredon (1968). In Nigeria, the mixing of Newcastle disease (Komarov) vaccine and fowl typhoid vaccine is routinely practised. The incidence of Newcastle disease (ND) and fowl pox (FP) is high (Hill and Davis, 1962; David-West, 1972). There is insufficient data concerning fowl typhoid (FT) but recently several outbreaks in some states of Nigeria in unvaccinated birds indicate that this infection may also be widespread. In French speaking West Africa where the same disease conditions are prevalent, Provost and Borredon (1968), successfully instituted a mixed vaccine, "Polyvia", against Newcastle disease, fowl pox and fowl typhoid.

The production and use in Nigeria of a similar vaccine incorporating the LaSota strain of NDV, Beaudette strain of FPV and Weybridge strain of FT is described herewith.

MATERIALS AND METHODS

Vaccine Production

(a) ND Vaccine (NDV)

Fertile 10 days old eggs were inoculated intra-allantoically with 0.1 ml of a 1 in 1000 dilution of seed virus with a titre of at least 10^4 EID_{50}/0.1 ml. After 3 days incubation at 37°C, viable eggs were spot-tested for the presence of virus by mixing a loopful of allantoic fluid with a drop of 10% washed chicken erythrocytes and examining them for agglutination. Positive amnio-allantoic fluids were harvested, pooled and mixed with an equal volume of stabilizer containing peptone, mannitol and buffer salts and stored at 4°C until tests for bacterial and fungal contamination were complete.

(b) FP Vaccine (FPV)

Fertile 12-day old eggs were inoculated with 0.2 ml of a 1 in 1000 dilution of seed virus containing 10^5.5 EID_{50}/0.1 ml, on the dropped chorioallantoic membrane and incubated at 37°C. Viable eggs after 5 days of incubation, were cut open and membranes harvested in small pools in universal bottles and stored at −20°C while sterility tests for bacterial and fungal contamination were carried out. Those pools with contamination were discarded and only sterile ones were ground with equal parts (v/v) of stabilizer and filtered through muslin cloth.
(c) *FT Vaccine*

Seed ampoules of attenuated Weybridge (smooth) strain of *Salmonella gallinarium* were inoculated into nutrient agar and then plated onto blood agar. A subculture in broth was made from pure colonies on blood agar and 24 hours later 5 ml of broth culture was seeded on to each of several Roux bottles containing solid medium made up of meat and yeast extract, peptone, glucose, sodium chloride and agar. After 48 hours of incubation at 37°C each bottle was examined for purity of growth and the organisms were decanted with 10 ml of stabilizer per bottle.

(d) *Freeze Drying of Vaccine*

One part of FPV, and two parts each of NDV and FT by volume were mixed and dispensed in 2.5 ml quantities per ampoule. No antibiotics were added. Primary drying was done on an Edwards EF 6 machine for 16 – 18 hours and after constriction, secondary drying was carried out for a further 12 to 14 hours. Ampoules were sealed under vacuum. Each ampoule contained 100 field doses. Ampoules were stored at -20°C until issued.

**Antibody Estimations**

Haemagglutination inhibiting (HI) antibodies to NDV were estimated using doubling virus dilutions against a constant 10-fold dilution of serum (alfa procedure: H.M.S.O. 1971). Agglutinating antibodies to *Salmonella pullorum* were detected by the rapid whole blood test using stained antigen (National Academy of Science, 1971). The response to FPV was tested only by challenge of vaccinated birds with virulent virus.

**Vaccine Assessment**

The vaccine was tested for safety, stability and potency as follows:

**Safety Test**

This was carried out using 50 one-day-old chicks, each inoculated intramuscularly (i/m) in the thigh with one field dose of the combined vaccine and observed frequently for the following three weeks. Further, 10 six-week old chicks were each inoculated with 10 field doses by the same route and observed for a similar length of time.

**Stability Test**

An accelerated stability test was performed by incubating a group of ampoules at 37°C for 7 days and then determining the titre of each component of the vaccine. Two ampoules were reconstituted to volume in an antibiotic concentrate containing penicillin and streptomycin to eliminate typhoid organisms and subsequent ten-fold dilutions were made in cold phosphate buffer solution (Dulbecco) over a range of 10^{-1} to 10^{-12}.

For NDV, 6 ten-days old fertile chicken eggs were each inoculated intra-allantoically with 0.1 ml of the 10.8 to 10^{-12} dilutions. After three days incubation at 37°C eggs were chilled and spot-tested for the presence of virus as described above. The fifty per cent end point was calculated by the method of Reed and Muench (1938).

The titre of FPV was determined using 6 twelve-days old fertile, chickens eggs each inoculated on the chorio-allantoic membrane with 0.1 ml of the 10^{2} to 10^{6} dilutions of the vaccine. After five days incubation, membranes
were examined for pock lesions and fifty per cent end points calculated.

The Fowl Typhoid content was titrated using a series of ten-fold dilutions of the vaccine made in cold normal saline ranging from $10^{-1}$ to $10^{-10}$. Using a pipette (25 drops per ml.) one drop was placed on a blood agar plate. Separate pipettes and plates were used for each dilution from $10^{-6}$ to $10^{-10}$. After 48 hours of incubation a viable count was made by enumerating colonies.

(c) Potency Tests and Field Trials

Unless otherwise mentioned all trials were undertaken in White Leghorn chicks from a closed flock of healthy birds known to be sero-negative to NDV and FT.

The same 50 one-day-old chicks used as part of the safety testing schedule were also used to assess serological response. Ten days after vaccination all birds were tested serologically for immunity to *S. pullorum*. Antibodies to NDV were measured at 3 and 4 weeks of age.

Thirty six of a group of 48 six-weeks old birds also received a single field dose by the i/m route and their sera tested for agglutinating FT antibodies and NDV HI antibodies at 21 days post-vaccination. At this time birds were divided into three groups, each containing 12 vaccinated and four unvaccinated individuals. Each group was challenged with a different valency of the trivalent vaccine.

Double vaccination was assessed by vaccinating a group of 50 one-day-old chicks, 24 of which were Rhode Island Reds and the remainder White Leghorns, and revaccinating them at seven weeks of age. NDV and FT antibody responses were measured as described above. These birds were not challenged.

The duration of immunity to combined vaccine was examined in 70 birds doubly vaccinated at 4 and 8 weeks of age with one field dose per bird. Serum samples were collected periodically and tested for NDV and FT antibodies and at varying intervals between 3 and 45 weeks post primary vaccination. Groups of 3 vaccinated birds and one unvaccinated control were challenged.

9309 chicks at Oke Afa Farms, Lagos, were vaccinated at 4 and 20 weeks of age (Fadeyi 1976) and some were challenged at the end of their laying period (73 weeks after primary vaccination). 100 chicks were vaccinated at 4 and 20 weeks at N.L.M.A. Poultry farm, Jos, and a few were challenged for ND at 26 weeks post primary vaccination. Out of 297 White Leghorn chicks 150 were vaccinated with the combined vaccine at 6 and 20 weeks of age at the University of Ibadan (Adene and Hill, 1976) and the remainder with conventional vaccine such as Hitchner B1 and Komarov for comparison on the productivity and performance.

**RESULTS**

Safety Tests

No abnormal clinical reactions were observed either in the group of day-old chicks receiving one field dose of vaccine, or in the six-weeks old birds receiving 10 field doses.

Stability Test

The residual titres in ampoules of freeze-dried vaccine maintained at 37°C for one week were significantly higher than those required by international standards (Table 1), and indicated that shelf lives of 7 days at room temperature, one year at 4°C or two years at -20°C, could be anticipated.
Table 1: Stability of Combined Vaccine

<table>
<thead>
<tr>
<th>Fraction of vaccine</th>
<th>Minimum required</th>
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<th>After 7 days at 37° C</th>
<th>After 2 years at −20° C</th>
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<td>FT</td>
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Figures as log_{10} per field dose.

Serological Response to Vaccine

In all experiments whole blood samples from chicks at 10 days of age agglutinated stained *S. pullorum* antigen.

The HI antibody levels to NDV at 3 and 4 weeks following a single intramuscular inoculation of one field dose at hatching are shown in Table 2. The White Leghorn chicks which were vaccinated twice showed NDV antibodies responses similar in titre to those described above, while Rhode Island Red chicks gave a mean level seven-fold higher probably because of the difference in body weights and development of hossa of Fabricius. The differences in mean antibody between these two breeds narrowed following revaccination at 4 weeks of age.

Birds vaccinated at 4 and 8 weeks of age developed a faster and higher antibody response to NDV. Antibody levels remained high during the five week period following revaccination but declined sharply thereafter; there was no difficulty however in demonstrating circulating antibodies up to the 35th week of this trials.

Challenge

Results of challenge tests are summarised in Table 3.

It was noted that birds having NDV HI titres of as low as 40 resisted challenge with virulent ND virus, and that birds surviving challenge showed an anamnestic response to this virus.

Challenge with fowl typhoid was considered one of the criteria of overt disease and survivors at day 21 were scored for lesion and re-isolation of virulent *S. gallinarum* (Williams Smith, 1955). With two doses the duration of immunity was observed up to 43.5 weeks.

DISCUSSION

From the results of the challenge tests and studies on performance of birds at N.V.R.I., Vom, N.L.M.A., Jos, University of Ibadan and Oke Afa Farm, Lagos, it is apparent that all 3 valencies were potent without any evidence of cross interference. The vaccine was found to be safe even for one-day-old chicks. Two doses at 4 and 20 weeks gave protection up to 73
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Table 2: Results of Haemagglutination Inhibition (HI) Tests Against Newcastle Disease Virus Using Alpha Procedure

Number of birds with Reciprocal HI titre

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Weeks post pli

Mary Vaccination

Weeks of chicken

Experiment No.
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<th>Weeks</th>
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Table 3: Periodic immunity challenge of birds vaccinated simultaneously with combined vaccine
weeks against ND & FP but not FT for which immunity lasted 43.5 weeks only. Smith (1969) recorded immunity to FT vaccination of only 34 weeks duration using a rough strain vaccine. Nevertheless vaccination is considered beneficial. Even in Europe poultry breeders are increasingly using vaccine for the control of Salmonellosis (Anon, 1978). In Nigeria, like many other developing countries, blood testing for Salmonellosis is voluntary. Therefore, the use of the smooth strain is still justifiable.

The Beaudette strain of FPV used has been universally applied elsewhere as the standard vaccine strain. The selection of the NDV strain, however, required careful consideration. The enzootic Nigerian strain is of the velogenic viscerotropic type (Kaschula 1961), David-West 1972, Nawathe et al. 1975). Mesogenic strains like Komarov, Roakin, Beaudette 'C', Mukteswar or Bnkowski confer solid immunity for over one year (Reeve et al. 1974). However, many workers are reluctant to advise their universal use because of side reactions (Winterfield and Fadly 1973, Allan and Dawson 1973, Higgins 1971 Gupta and Rao 1959). There appears to be no ideal NDV vaccine strain.

We have selected the LaSota strain because among the lentogenic strains, it is considered to be superior to Hitchner B1, Ulster 2C, F (Asplin) etc. (Spalatin and others 1976, Owolodun 1968, Roepke 1973, Anon 1971b). Thorne and MacLeod (1960), found the 'F' strain to be unsuitable in Nigeria because of its neuropathogenicity, although it is widely used in the Middle East and the Far East (Chu & Rizk 1972, Higgins 1971). Furthermore experience in Nigeria had shown the LaSota vaccine to confer immunity for 6 months when administered at 3 weeks of age by drinking water route (Owolodun & Ajiboye 1975).

ACKNOWLEDGEMENT

The authors wish to express their gratitude to Professor D.H. Hill, Dr. D.F. Adene, Dr. J. C. A. Ajubo and Dr. A. A. Fadeyi for conducting experiments on their farms and the Director, National Veterinary Research Institute, Vom, for providing facilities and permission to publish this paper.

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Received for publication on 21st March, 1980
EQUINE LYMPHOSARCOMA IN THE SUDAN

M.H. TAGELDIN, S.H. IDRIS and M. HERCEG,*
Veterinary Research and Administration, P.O. Box 8067, Khartoum, Sudan.

SUMMARY

Three cases of equine lymphosarcoma were described. Two cases, one in a horse and the other in a mare; the latter was infected with African horse sickness type 9, in addition to lymphosarcoma. The remaining case, in a female donkey; the lesion being confined to the perineal area.

INTRODUCTION

Although lymphosarcoma is common in dogs and cats (Cotchin, 1969) it is rare in equines (Runnels and Benbrook, 1944; Moulton, 1961; Conboy and Powers, 1971; Neufeld, 1973 and Sundberg et al., 1949) or in the intestine (Neufeld, 1973), from there it metastasises to different parts of the body. African horse sickness is a stationary diseases of equine in the Sudan. One horse, succumbed to this disease showing, in addition, lesions of lymphosarcoma; hence the following cases are believed to be of interest and worthy of report.

Case 1

A six year old indigenous female donkey was admitted to Singa Veterinary Clinic, with a swelling of about 5 cm in diameter in the perineal area. The animal was in fair condition and there were no apparent ill effects. The tumour mass was removed surgically and fixed in 10 percent formal saline.

Microscopically, the lymphocytes were pleomorphic and mitotic figures were moderate.

Case 2

A three year old cross-breed horse was brought to Khartoum Veterinary Clinic showing respiratory symptoms (harried respiration and cough) and had no appetite for three days. Clinical examination revealed anorexia and crippling of both lungs. Pneumonia was diagnosed and antibiotics were administered. The animal deteriorated and subsequently died after two days.

On post-mortem examination the lungs were oedematous. There was hydrothorax. The mesenteric lymph nodes were grossly enlarged measuring 12 x 10 x 7 cm. Small nodules the size of chick peas were observed on the serosal surface of both small and large intestines. The intestinal contents was watery and the mucosa was slightly thickened. The liver was slightly enlarged.

Histopathologically: Focal infiltration of lymphocytic and lymphoblastic cells were seen along the small and large intestine. The tissues of mesenteric lymph nodes were replaced by tumour cells. The liver, spleen, kidney and other organs showed no tumour infiltration.

Case 3

A six year old cross-bred mare was submitted to Khartoum Veterinary Clinic showing respiratory distress. On examination, there was oedematous swelling of the supraorbital Fossa.

*Department of Pathology, Faculty of Veterinary Science, Zagreb University, Yugoslavia.
The conjunctival and nasal mucous membrane were severely congested. The animal died shortly afterwards. Necropsy performed revealed the following:

The striking features on post-mortem examination were hydrothorax, hydropericardium and oedematous infiltration of both lungs. Trachea and bronchi were filled with an abundant amount of yellowish foamy fluid. The bronchial lymph nodes were enlarged.

Petecheal haemorrhages on the epicardium and whitish streaks on the heart muscle were observed. The intestinal wall was thick with greyish nodules and a bacon-like consistency. There were a few haemorrhagic ulcers which contained necrotic debris. The intestinal contents was watery and tinged with blood. Mesenteric lymph nodes were enlarged creating, here and there, large conglomerates. The remaining lymph nodes of the body were unchanged. The liver was hyperemic. The spleen, kidney and other organs were macroscopically unchanged.

Specimens were taken from the spleen and inoculated into suckling white mice. African horse sickness virus was isolated and identified as type 9 (Eisa, 1974).

Histopathology

The cell population of the mesenteric lymph nodes and intestine were pleomorphic. It consisted mostly of cells of the lymphoid and lymphoblastic types. Here and there neoplastic cells resemble reticulum cells in appearance and order; but because of numerous mitotic figures, they lost their ability for further differentiation. The neoplastic cells infiltrated all layers of intestinal wall including serosa. Examination of the heart revealed, small clusters of neoplastic cells resembling lymphoblast infiltrating between myocardial fibres. The lungs, liver spleen, peripheral lymph nodes, central nervous system and other organs were without tumour growth.

DISCUSSION

The three cases above are reported in young animals (3-6 years), while findings by Moulton (1961) and Sundbergs' et al., (1977) indicated that lymphosarcoma usually affects older animals from 9 years onwards. Our findings suggest that young animals could similarly be affected. This is in agreement with the finding of Neufeld (1973). In case 3 the association of lymphosarcoma with African horse sickness, provides a special interest. Since lymphosarcoma is a chronic disease involving reticular tissue, the body cellular defensive mechanism is greatly lowered, thus it seems logical that AHS in case 3 was a secondary complication.

ACKNOWLEDGEMENTS

We are grateful to Dr. A.M. Shommein, Head Department of Pathology, for reading and revising the manuscript. Thanks are also due to Dr. El Amin Daffalla for the specimens sent from Singa.

REFERENCES

RINGWORM CAUSED BY TRICHOPHYTON VERRUCOSUM IN YOUNG GOATS: A CASE REPORT

C.N. CHINEME, J.O. ADEKEYE and S.A. BIDA
Department of Veterinary Pathology and Microbiology, Ahmadu Bello University, Zaria, Nigeria.

SUMMARY

An outbreak of ringworm caused by Trichophyton verrucosum in a goat herd is described. All ages of goats were affected the disease being most severe in younger ones between the ages of 3 and 5 months. Clinical signs shown were mainly intense scratching at the wood pens and unthriftness. None of the sheep kept in the same house with the goats showed any clinical signs or lesions. Gross lesions were consistently present in the affected goats and were characterized by areas of varying sizes showing scaly and alopecic eruptions in various parts of the body but most predominantly on the face and pinna. Diagnosis of trichophytosis was based on positive culture of T. verrucosum from skin lesions. Oral administration of griseofulvin at dosage rate of 25 mg per kilogram per day per os for 21 days cleared the skin lesions in all the goats after 6 weeks.

INTRODUCTION

Several species of the genus Trichophyton have been found in a variety of domestic and wild animals in many parts of the world. Trichophytosis which may be characterized by a wide variation of clinical signs is commonly caused by T. verrucosum in cattle (Jungerman & Schwartzman 1972, Buxton and Fraser 1977); T. mentagrophytes in dogs, cats and horses; and T. equinum in horses (Jungerman and Schwartzman, 1972). Spontaneous trichophytosis in goats is extremely rare (Jungerman and Schwartzman, 1972), and documented reports of the disease in the Nigerian indigenous goats are apparently non-existent. This communication therefore reports the occurrence of trichophytosis caused by T. verrucosum in young goats in Nigeria.

HISTORY OF OUTBREAK

In March 1979, 3 goats in a herd of 36 showed areas of alopecia and encrustations on the pinna. Within approximately 7 days, the lesions had spread to other areas including around the ears, horns, and eyelids, at the root of the nose and around the tail (Fig. 1). Later, confluent lesions were seen on the flanks also (Fig. 2). Similar lesions were seen also on 12 more goats in the herd. All the goats the ages of which ranged from approximately 3 to 8 months were bought from local markets in and around Zaria about four weeks prior to the appearance of lesions. They were being housed singly in partitioned pens, some of which housed 30 sheep also. There was no mortality during the period of outbreak.

The animal attendant who looked after the goats and sheep developed lesion on his right arm during the outbreak but it healed after 7 days topical application of iodine liniment.

MATERIALS AND METHODS

Following the initial tentative diagnosis in the first 3 goats, follow-up studies
Fig. 1. Goat showing skin lesions of ringworm caused by *T. verrucosum* of approximately 7 days duration.

Fig. 2. Goat showing long-standing confluent skin lesions of Trichophytosis.
were conducted to isolate the specific aetiologic agent by the culture method. Skin scrapings and hair samples from and around the lesions from each of the affected goats were submitted for fungal culture and isolation. Hairs from each sample were examined directly under light microscope after clearing on a slide with 10% potassium hydroxide solution. Part of each sample was inoculated into 2 test tubes of DIFCO mycobiotic agar slants, incubated at room temperature and examined daily for fungal growth. Nutritional tests for the differentiation of Trichophyton species were then carried out on the fungal growths as described by Georg and Camp (1957). Direct mounts of growths from the agar slants were stained with lactophenol cotton blue and examined microscopically.

Each of the affected goats was given 25 mg/kg body weight of griseofulvin per os per day for 21 days.

RESULTS

Direct microscopic examination of the digested hairs showed fungal organisms that were characteristic of Trichophyton species. Slow growing fungal colonies ranging from 5 – 15 mm in diameter were recovered in culture after 12 days. The colour was off-white to light yellow with scanty aerial mycelia which were heaped and folded. A slide culture of the fungus on DIFCO potato dextrose agar produced chlamydospores arranged in long chains. Favic chandeliers were seen but no macroconidia and microconidia were present. The nutritional requirements of the fungus were consistent with those of Trichophyton verrucosum as described by Wolf, Russel and Shimoda (1975).

After 5 days of treatment, an initial change in the character of the skin lesions was seen. The powdery crusty lesions had started to disappear and by the second week of treatment, those lesions had disappeared totally leaving bare alopecia only. After approximately 3 weeks of treatment there was initial re-growth of hairs in the alopecic areas of the skin. By the sixth week there was a complete and normal regrowth of hairs and no gross lesions were detected on the skin of any of the goats. The rest of the goats and all the sheep housed with the diseased goats showed no visible skin lesions throughout the period of the outbreak.

DISCUSSION

Primary attacks by T. verrucosum have mainly been recorded from cattle and infection of other animals is uncommon and occurs only in connection with infected cattle (Dvorak and Otcenasek, 1969). In the present outbreak, the goats were not housed with cattle, and cattle could not have been kept in the goat pens previously because the pens were too small to house cattle. Infection therefore could not have originated from dried arthrospores that could have remained for years in buildings which had been contaminated by the rubbing of infected cattle (Mortimore, 1955).

As soon as the goats were brought to the farm, they were kept singly in separate pens. Contact between them was therefore minimal once on the farm. Consequently the spread of infection could not therefore be satisfactorily ascribed to contact between goats on the farm. The feed could be implicated as a common vehicle of transmission especially as most lesions started on the facial area; however the sheep and the unaffected goats which were being given the same feed as the diseased goats did not contract the disease. The goat at-
tendant could not be implicated as a common source of spread since he became infected later than the goats. The history of the goats showed however that the local supplier bought them from various farmers who usually raise cattle, sheep and goats together. It is therefore possible that some of the goats raised along with cattle could have had non-clinical trichophytosis at the time of their purchase and in the farm developed clinical skin lesions and thus transmitted the organisms to other goats.

The high morbidity among the younger goats in this outbreak simulates the high infectivity rate of the organism for cattle in which on occasional farms in England, it was common to find that all the calves were affected (La Touche, 1955). The unthriftness of the affected goats however is not a common feature of the disease in cattle. Only in severely affected calves is there any evidence of retarded growth (Mortimore, 1955).

The importance of the use of griseofulvin in the treatment and subsequent disappearance of lesions in the affected goats cannot be well appreciated and may not be of much significance because the majority of lesions in valves also heal spontaneously without treatment (Mortimore, 1955). Subsequent infection of the goat attendant by T. verrucosum however emphasizes the zoonotic importance of the fungus. The basis for a rational use of expensive fungicides in domestic food animals infected by dermatophytes might be found only in preventing the transmission of the fungi to people. This outbreak highlights the dangers inherent in the purchase of young animals for experiment or otherwise from various markets or farms without knowing the history of the parent stock and management practices. Such animals may be carrying or incubating such diseases as may have devastating effects when they are mixed with other animals.

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Received for publication on 4th March, 1980
THE INCIDENCE AND SEROTYPES OF GROUP B-STREPTOCOCCI IN DAIRY CATTLE

M. T. A. SHIGIDI and I.E. MAMOUN,
Faculty of Veterinary Science, P.O. Box 32, Khartoum North, Sudan.

SUMMARY

Bacteriological examinations were made on composite quarter milk samples from cows in six herds. *Streptococcus agalactiae* was isolated from all herds. Single samples were tested and the sample incidence was 31.4%. Twenty-five to 39% of the isolates were resistant to penicillin, cloxacillin, tetracycline and streptomycin. Types II and Ia were the most commonly encountered group B serotypes. Cross-infection between man and animals must be kept in mind.

INTRODUCTION

*Streptococcus agalactiae* has long been recognized as a major cause of mastitis in cattle (Stableforth, 1959). More recently it has been isolated from cases of septicaemia and meningitis in humans (MacKnight et al., 1968; Baker et al., 1973; Paker, 1979). There appears to be a difference of opinion as to whether the bovine and human strains are distinct. Butter and De Moor (1967) and Norcross and Oliver (1976) found some physiological and serological differences between the two populations. However, Hahn (1977) noted that the bovine and human strains were similar in pathogenicity and in cultural, biochemical and serological characteristics and that cross-infection between cattle and man might occur.

Group B streptococci were subdivided by the precipitin test into types designated I, II and III on the basis of their polysaccharide antigens (Lancefield, 1934). Based on their polysaccharide antigens and minor protein determinants, type I strains were subsequently separated into types 1a, 1b and 1c by the precipitin and mouse-protection tests (Lancefield, 1938; Wilkinson and Eagon, 1971). The only reported attempt to serologically classify B-haemolytic streptococci from cattle in the Sudan was that of Bagadi (1970). Of 78 strains that he classified, 15 belonged to group B, but their serotypes were not determined. The aim of the present study was to determine the incidence and types of group B streptococci isolated from dairy cattle in different parts of the Sudan.

MATERIALS AND METHODS

Source of Organisms

Streptococci were isolated from composite quarter milk samples taken at random from 373 milking cows in seven farms. The herds were located in the University farm, Kuku and Kaffory (Khartoum Province); Obeid (Northern Kordofan Province); Atbara (Nile Province); Nesheisheiba (Gezira Province). Animals were sampled on only one occasion and samples were streaked on azide blood agar base (Oxoid CM 259) containing 5% sheep blood. Plates were incubated overnight at 37°C. Colonies typical of streptococci were identified by biochemical reactions and precipitin tests. Reference strains of group B streptococci 090R, 090, H36B, A909, 18RS21 and D136C
representing group B and types 1a, 1b, 1c, II and III respectively were obtained from the National Institute of Public Health, Bilthoven, the Netherlands.

Biochemical tests

The CAMP reaction was determined on sheep blood agar plates streaked with a strain of *Staphylococcus aureus* producing B-haemolysin (Christie, Atkins and Munch-Petersen, 1944). Fermentation of carbohydrates, hydrolysis of aesculin, arginine and hippurate and growth on 40% bile were performed as described by Cowan and Steel (1975).

Sensitivity to antibiotics

This was determined by the disc-diffusion method. From a 24-hour growth in Todd-Hewitt broth (Oxoid CM189), 0.025 ml was spread on Muller-Hinton agar (Oxoid CM331) plates. These were overlaid by commercial sensitivity discs containing ampicillin (2 mg), bacitracin (5 units), chloramphenicol (10 mg), clindamycin (2 mg), cloxacinil (1 mg), erythromycin (10 mg), lincomycin (2 mg), methicillin (5 mg), neomycin (30 mg), penicillin (1.5 units), streptomycin (10 mg), and tetracycline (10 mg). After incubation overnight at 37°C, strains were classified as sensitive, moderately sensitive or resistant by comparing the diameters of the zones of inhibition of growth with those produced on the sensitive *Staph. aureus* Oxford strain NCTC6571 as described by Cruickshank et al., 1975).

Preparation of antisera

Rabbits were immunized intravenously with vaccines prepared from the reference strains. The organisms were grown in Todd-Hewitt broth at 37°C for 18 hours and killed by heating at 60°C for 30 minutes. The cells were washed three times in physiological saline. Standard suspensions were prepared by resuspending the washed organisms in saline to give an optical density of 0.7 at 530 nm. The sterility of the vaccines was checked by streaking on to blood agar plates. Immunization schedules and absorption of antisera were carried out as described by Lancefield, McCarty and Everly (1975). Heat-killed cultures of types 1a and 1c were used to absorb antisera against types 1c and 1b respectively, the remaining antisera were used unabsorbed.

Preparation of antigens

A single colony of each streptococcus was transferred into 40 ml Todd-Hewitt broth and incubated for 18 hours at 37°C. The growth was sedimented by centrifugation at 1500 g and extracted with 0.4 ml of N/5 HC1. The extract was neutralized with N/5 NaOH using phenol red as indicator. After a final centrifugation, the supernatant was used as antigen for both the gel diffusion and counterimmunoelectrophoresis tests.

Serological tests

Antigens extracted from the isolates were first tested with group B antiserum by the agar gel diffusion test on microscope slides. The gel consisted of 1% Noble agar (Difco) in 0.05 M barbitone acetate buffer, pH 8.6. The antigens and antiserum were placed in wells 4 mm in diameter and 7 mm apart and the slides were examined for lines of precipitation after 24 and 48 hours in a moist chamber at room temperature. Cultures which reacted with group B antiserum were tested against the type specific antisera by the counterimmunoelectrophoresis test described by Edwards and Larson (1973). The test was carried out
on microscope slides covered with 1% agarose dissolved in the same buffer used for the gel diffusion test. Electrophoresis was run at a constant voltage of 6 volts per centimetre for 1 hour at room temperature.

**RESULTS**

*Str. agalactiae* was isolated from 117 of the 373 cows examined, giving a cow incidence of 31.4%. The prevalence varied from 24.4% in one herd to 46% in another (Table 1).

The strains showed some variation in physiological properties. Only 45% out of the 117 isolates had a clear narrow zone of B-haemolysis. Nine strains (10.5%) were CAMP test negative and 35 strains (41%) were not inhibited by 40% bile. Fifteen strains (17.6%) failed to ferment lactose, 2.5% failed to ferment trehalose and 27% failed to ferment salcin. All strains fermented maltose and sucrose while none fermented raffinose, mannitol, sorbitol or inulin. All strains hydrolysed sodium hippurate and produced ammonia from arginine while none split aesculin.

Results of antibiotic sensitivity tests are summarized in Table 2. It was found that there was variation in susceptibility among isolates. However, the only antibiotics for which there were significant populations of resistant strains were cloxacillin, penicillin, neomycin, streptomycin and tetracycline.

All the strains could be serotyped. Table 1 gives the serological types found among the 117 strains from different herds. Type II was the most frequently encountered (49.6%) followed by type 1a (35%). Types 1b, 1c and III were much less frequent with an incidence of 6.9%, 3.4% and 5.1% respectively. The distribution of serotypes in different herds did not differ significantly, however, types 1b, 1c and III were not detected in the University of Khartoum and Kuku herds.

### Table 1: Incidence and types of *Str. agalactiae* in dairy herds

<table>
<thead>
<tr>
<th>Herd</th>
<th>No. of cows examined</th>
<th>No. of cows with <em>Str. agalactiae</em></th>
<th>No. 1  with</th>
<th>str. agalactiae type</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>13(27.7)*</td>
<td>la</td>
<td>1b</td>
</tr>
<tr>
<td>University of</td>
<td>47</td>
<td></td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Khartoum</td>
<td></td>
<td></td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Kuku</td>
<td>43</td>
<td>15(34.9)</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Kaffory</td>
<td>50</td>
<td>23(46.0)</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Obeid</td>
<td>90</td>
<td>28(31.1)</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Atbara</td>
<td>53</td>
<td>16(30.2)</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Nesheisheiba</td>
<td>90</td>
<td>22(24.4)</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>373</strong></td>
<td><strong>117(31.4)</strong></td>
<td><strong>41(35.0)</strong></td>
<td><strong>8(6.9)</strong></td>
</tr>
</tbody>
</table>

*Figures in brackets are percentages*
Table 2: Sensitivity of 117 strains of *Str. agalactiae* to antibiotics

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Concentration in µg</th>
<th>No. sensitive strains</th>
<th>No. moderately sensitive strains</th>
<th>No. resistant strains</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>2</td>
<td>65(55.6)</td>
<td>52(44.4)</td>
<td>0(0)</td>
</tr>
<tr>
<td>Bacitracin</td>
<td>5*</td>
<td>99(84.6)</td>
<td>12(10.3)</td>
<td>6(5.1)</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>10</td>
<td>107(91.5)</td>
<td>6(5.1)</td>
<td>4(3.4)</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>2</td>
<td>63(53.9)</td>
<td>48(41.0)</td>
<td>6(5.1)</td>
</tr>
<tr>
<td>Cloxacillin</td>
<td>1</td>
<td>51(43.6)</td>
<td>30(25.6)</td>
<td>36(30.8)</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>10</td>
<td>73(62.4)</td>
<td>40(34.2)</td>
<td>4(3.4)</td>
</tr>
<tr>
<td>Lincomycin</td>
<td>2</td>
<td>61(52.1)</td>
<td>50(42.8)</td>
<td>6(5.1)</td>
</tr>
<tr>
<td>Penicillin</td>
<td>1.5*</td>
<td>42(35.9)</td>
<td>45(38.5)</td>
<td>30(25.6)</td>
</tr>
<tr>
<td>Neomycin</td>
<td>5</td>
<td>84(71.8)</td>
<td>15(12.8)</td>
<td>18(15.4)</td>
</tr>
<tr>
<td>Streptomycin</td>
<td>10</td>
<td>56(47.9)</td>
<td>15(12.8)</td>
<td>46(39.3)</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>10</td>
<td>71(60.7)</td>
<td>16(13.7)</td>
<td>30(25.6)</td>
</tr>
</tbody>
</table>

*Units
Figures in brackets are percentages

**DISCUSSION**

The results show that *Str. agalactiae* is widespread in the udders of dairy cows in many herds in the Sudan. The isolation of the organism from 31% of the cows was higher than the 16% reported by Bagadi (1970) from the same herds. Despite the very low standard of hygiene in all the farms, the cow incidence was similar to the 36% reported in Italy by Valenti and Caldora (1974) and to the 33% reported by Pearson, Pollock and Greer (1979) in Northern Ireland. However, the detection rate might have increased in this study by repeated sampling of cows and by not using a selective medium for primary isolation. The incidence of carriers would undoubtedly be lowered if more efforts were made to improve hygiene together with mass treatment of cows during the dry period.

The biochemical characteristics obtained for the isolates were similar to those described for *Str. agalactiae* (Cowan and Steel, 1975), but a proportion of strains gave negative CAMP tests and failed to ferment lactose and salicin. Similar variations were noted by Elliott, Tatterfield and Brookbansk (1976) and Norcross and Oliver (1976).

Isolates of *Str. agalactiae* were either sensitive or moderately sensitive to most antibiotics tested which agrees with the results of other workers (Anthony and Concepcion, 1975; Havelka, 1976). Butter and De Moor (1967) and Norcross and Oliver (1976) noted that *Str. agalactiae* of bovine origin tended to be more sensitive to bacitracin and tetracycline than the human strains. In this study the majority of strains were susceptible to bacitracin, but a significant population of strains was resistant to tetracycline. The occurrence of resistant strains to cloxacillin, penicillin, neomycin and tetracycline is of clinical significance, since these drugs are often used to treat streptococcal infections in cattle. There was no correlation between antibiotic susceptibility pattern, source of culture or serotype.

All isolates were typable with the sensitive counter-immunoelectrophoresis technique. The majority of strains (50%) belonged to type IIA followed by type Ia (35%), whereas types 1b, III and 1c
(in decreasing order) were less frequent. The distribution of the various types in the Sudan agrees favourably with that of bovine group B strains isolated by Butter and De Moor (1967) in the Netherlands and by Haug (1972) in Norway. They found type II predominant and constituted 44% and 55% respectively of their typable strains. However, it is possible that the distribution of serotypes may vary in different countries, since Norcross and Oliver (1976) identified type 1a as the most common type in cattle in New York State, whereas Stableforth (1937 and 38) concluded that types 1a and 1b did not occur in cattle in Britain.

*Str. agalactiae* has recently been recognized as a significant human pathogen. Types 1a and III are the two types most frequently isolated from systemic infections in neonates and occasionally in adults. Type II is most frequent in superficial infections (Parker, 1979). Infants usually obtain the organism from their mother’s vagina, but the epidemiology of infections in adults is not known. Studies show that the bovine and human strains of *Str. agalactiae* have overlapping characters and that cross-infection between man and cattle may occur (Hahn, 1977). There is no record of the incidence and types of group B streptococci associated with human infections in the Sudan. The standard of hygiene in dairy farms is poor. Animals are hand milked and a proportion of milk is consumed without pasteurisation. The risk of spread of the organism and cross-infection between man and animals is therefore high.

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*Received for publication on 9th April, 1980*
PATHOLOGICAL STUDIES ON SHEEP AND GOATS PNEUMONIA IN THE SUDAN

Part II. Experimental Infection

M.I. ABUBAKR, M.E. ELFAKI, S.A. ABDALLA and S.M. KAMAL,
Veterinary Research Administration, P.O. Box 8067, Alamarat, Khartoum, Sudan.

SUMMARY

A total of 35 sheep and 28 goats were intratracheally infected with Staphylococcus aureus, Staptococcus sp. Proteus mirabilis, Pseudomonas aeruginosa and Mycoplasma arginini, which were isolated from pneumonic lungs of naturally diseased sheep and goats. Five sheep and four goats served as controls.

The observation and reading of clinical signs, pathological lesions of the disease and also microbiological studies were done.

The inoculated organisms were reisolated from most of the experimentally infected animals.

Bronchopneumonia, fibrinous pneumonia and chronic interstitial pneumonia were experimentally produced.

INTRODUCTION

In our previous communication (Abubakr et al., 1979) naturally acquired sheep and goats pneumonia was studied in three provinces of the Sudan.

This communication describes experiments conducted to investigate the pathogenicity of Staphylococcus aureus, Streptococcus sp. proteus mirabilis, Pseudomonas aeruginosa and Mycoplasma arginini, inoculated intratracheally, in sheep and goats. It also describes the relationship the gross and histopathological lesions in the pneumonic lungs of experimentally infected sheep and goats bear to those of the naturally infected animals.

MATERIAL AND METHODS

A total of 35 sheep and 28 goats purchased locally from nomadic herds were used. They weighed between 20 and 25 kilograms and were about 3-5 years old.

These animals were divided at random into six experimental groups consisting of 5 sheep and 4 goats in each group and one control group consisting of 5 sheep and 4 goats.

The experimental and control animals were housed and kept under the same conditions and were clinically observed for two weeks before the start of the experiment. Body temperatures were taken twice daily during this period.

The experimental animals were intratracheally inoculated. Bacterial organisms were given as 10 ml of broth culture containing 3.5 x 10⁸ to 10⁹ colony forming units /ml. Mycoplasma arginini was given as 10 ml of broth culture containing 3.5 x 10⁹ colony forming units /millilitre. Each animal in the control group was intratracheally inoculated with 10 ml of sterile broth.

During the experimental period, body temperatures were taken twice daily, (morning and evening), and the animals were examined for nasal discharge, Coughing and any other signs of respiratory discomfort. Dead animals, animals slaughtered in a moribund state and those slaughtered at the end of the experiment were necropsied, gross lesions observed were recorded and material for pathological and microbiological
examinations were taken. The methods of collection and examination of these tissues were as described previously (Abubakr et al. 1979). Runnels, Monlux and Monlux method of classification was again used to classify the experimentally produced pneumonias (Runnels, Monlux and Monlux, 1965).

Clinical Symptoms

The normal body temperature of the animals ranged between $98^\circ$ F in the morning and $103^\circ$ F in the evening for 14 days before inoculation. For the first two days post-inoculation no clinical symptoms were detected.

On the third day post-inoculation the clinical symptoms started to appear. The main clinical symptoms were rise in body temperature, exceeding $104^\circ$ F, coughing, laboured respiration, and, in some cases, lack of appetite, depression (Fig 1) and recumbency (Fig 2).

Death occurred in 15% of the experimentally infected animals a moribund state developed in 13% of them and the rest (72%) were slaughtered six weeks after intratracheal inoculation.

Fig. 1. Depressed experimental animal

Fig. 2. Recumbent experimental animal
Pathological Findings

As in natural infection, gross lesions were equally distributed in both right and left sides of the lung affecting all lobes without any significant frequency. Nevertheless the lesions were usually encountered in the antero-ventral portions of the lobes (apical, cardiac and intermediate lobes) with apical lobe on each side being more extensively affected than the diaphragmatic.

Three types of pneumonia were observed in the affected animals. The gross and histological lung lesions were similar to those obtained in case of natural infection, except that they only differed in extent and severity. Experimentally produced lesions were less extensive and less expressed than those found in naturally diseased animals.

1. Bronchopneumonia

This type of pneumonia was encountered in sheep and goats inoculated with *Staphylococcus aureus* and *Streptococcus* sp. Twenty two percent of the infected animals died. 22% were slaughtered in a moribund state and 56% were slaughtered at the end of the experiment.

The pneumonia, in the early stages was lobular in distribution, but by the time death of the animal occurred, the lobar pattern of the affected areas had disappeared and large portions of the lobes or even entire lobes were affected.

The main gross pathological lesions were congestion, oedema, and emphysema of the lungs, (Figs 3 and 4) and hydropericardium. In single cases, red hepatization of the lung, hydroperitonitis, tracheal froth, adhesion of the pericardium to the chest wall, and kidney haemorrhages were encountered. As in case of natural infection histologically the bronchial lumina were full of mucous, leucocytes and desquamated cells (Fig 5.) The bronchial walls, the peribronchial tissue and adjacent alveolar septa were infiltrated with cells. The alveolar capillaries were dilated and engorged with blood.

Fig. 3. Bronchopneumonia. Oedematous and congested lung
Fig. 4. Emphysematous lung

Fig. 5. Bronchopneumonia. The bronchial lumen is filled with leucocytes, mucous and desquamated cells. H and E staining (X 100).
2. Fibrinous Pneumonia.

Fibrinous pneumonia occurred in three groups of infected animals namely:

a. Those infected with *Proteus mirabilis*,
b. those infected with *Pseudomonas aeruginosa*, and
c. those infected with a combination of these two bacteria and *Mycoplasma arginini*.

In group a, 22% of the infected animals died, 11% were sacrificed in a moribund state, and 67% were killed at the end of the experiment. In group b, 11% were sacrificed in a moribund state and, 67% were killed at the end of the experiment.

The main gross pathological lesions were congestion and oedema of the lungs, froth and haemorrhages in the tracheo-bronchial pathways, adhesions of the pleura to the chest wall (Fig 6), and hydropericardium. The overlying pleura was dull, red or red-black and haemorrhagic, a pseudomembrane was formed on the pleura in areas corresponding to the underlying pneumonia.

Microscopically the alveoli were invaded by fibroblasts resulting in areas of organization. Only few alveoli scattered through the newly formed connective tissue were seen (Fig 7). The pleura was covered by a film of fibrin and leucocytes (Fig 8).

3. Chronic interstitial pneumonia.

This type of pneumonia was shown by the group of animals experimentally infected with a combination of *Staphylococcus aureus*, *Streptococcus* sp. and *Mycoplasma arginini*. Twenty two percent of the infected animals died of the disease, 11% were sacrificed in a moribund stage, and 67% were slaughtered at the end of the experiment.

![Fig. 6. Fibrinous pneumonia. Note adhesion of the pleura to the chest wall.](image)
Fig. 7. Fibrinous pneumonia. Complete replacement of exudate by fibroblasts. H and E staining (X 100).

Fig. 8. Fibrinous pneumonia. Pleura covered by a film of fibrin and leucocytes. H and E staining (X 100).
The lungs showed inflammatory oedema, congestion and emphysema and the tracheobronchial pathways were haemorrhagic (Fig 9). There was hydropericardium. Microscopic examination indicated that the main lesions were diffuse alveolar oedema. The alveolar septa were oedematous and thickened with a cellular infiltration of neutrophils, monocytes and fibrin. The septal capillaries were congested. The alveolar lumina contained a proteinacious fluid and macrophages (Fig 10).

4. All the animals intratracheally inoculated with Mycoplasma arginini alone did not show any clinical signs of the disease.

Both bronchopneumonia and chronic interstitial pneumonia were coupled with diffuse alveolar emphysema, oedema and congestion. Distension of the alveoli and bronchioles, and knobs formation at the tips of the broken septa were evident (Fig. II). Atelectasis, as a complication of bronchopneumonia, was again encountered.

A virus, which is still under identification, was isolated from four sheep. Two of these animals were inoculated with Pseudomonas aeruginosa, one animal was inoculated with Mycoplasma arginini, and the fourth one was inoculated with a combination of Pseudomonas aeruginosa, Proteus mirabilis and Mycoplasma arginini.

All the animals in the control group were clinically normal. None of these animals showed macroscopic or microscopic pathological lesions in the lungs, (Fig. 12), or in any of the other vital organs.

Fig. 9. Chronic interstitial pneumonia. Note the tracheal haemorrhages.
Fig. 10. Chronic interstitial pneumonia. Note thickening of the alveolar walls, congestion of septal capillaries, and fluid in the alveolar lumina. H and E staining (X 100).

Fig. 11. Diffuse alveolar emphysema. Note the distended bronchioles and alveoli, and the knobs formed at the tips of broken septa. H and E staining (X 100).
DISCUSSION

In our previous communication, a justification for the selective involvement of the anteroventral portions of the lungs, exudation into the alveolar walls, and the occurrence of atelectasis as a complication of bronchopneumonia, was given (Abubakr et al., 1979).

The pulmonary congestion and oedema revealed in bronchopneumonia, fibrinous and chronic interstitial pneumonia reflects their high non-specificity and confirms the postulation of Ikede (1977) that pulmonary congestion and oedema apart from being a sequel to inflammation may also be predisposing to it.

We are of the opinion that the death of some of the experimentally infected animals was due to toxaemia or inadequate gaseous exchanges in the lungs or to cardiac failure in chronic cases.

The recovery of the inoculated organisms from some of the experimentally infected animals that remained clinically healthy is suggestive of clinically undetectable carrier state.

Studying the results obtained from naturally diseased and experimentally infected animals it could be concluded:

a. that many factors such as environmen
tal conditions, management and genetic status of the animal may influence the manner in which the animal reacts to respiratory infections.

b. that the various types of pneumonia encountered are probably the result of complex interactions between many factors rather than the result of specific infections.

c. that a considerable number of specific infectious agents may be involved in the disease, and that these agents
may be present alone, or in different combinations.
d. that the herd may be harbouring a
number of specific infectious agents
without showing any clinical signs
of respiratory disease.

ACKNOWLEDGEMENTS

We are greatly indebted to D. M.S.
Harbi, Dr. M.S. Mohamed, D. M.A.
Salah and Dr. M.A. Khalil for isolation
and identification of Mycoplasma argi-
nini. Our thanks are also due to Dr.
G.A. Azheri and Dr. M.A. Salih for
bacterial isolation and identification.
The authors with to express their
thanks to the Under Seceratry, Animal
Resources, for his permission to publish
this manuscript.

Our thanks are also due to Dr. A.M.
Shommein for his criticism and useful
comments. The assistance of A.A.
Mohammed in carrying out macro-
and microphotography, and the technical
assistance of our technicians in the
different departments taking part in
this work is highly appreciated.

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Received for publication on 17th December, 1979
THE ERADICATION OF CONTAGIOUS BOVINE PLEUROPNEUMONIA IN NIGERIA: PROSPECTS AND PROBLEMS

T.L.O. OSIYEMI,
National Veterinary Research Institute, P.O. Box 38, Vom, Nigeria.

SUMMARY
The prevalence of contagious bovine pleuropneumonia in Nigeria is of very low order. The incidence of the disease has been reported at various times. From previous years of zero incidence, it rose to 15% by 1970 and started declining in the subsequent years as a result of control measures. It is proposed that control could be achieved within a decade by planned programme of mass vaccination.

INTRODUCTION
Contagious Bovine Pleuropneumonia (CBPP) is an eradicable disease of cattle caused by Mycoplasma mycoides var. mycoides (Hudson 1971). The disease had plagued cattle in Europe and elsewhere during the 19th century, as well as in the early decades of the 20th century. Its eradication at that time was achieved through slaughter policies (McFadyean 1925). The disease was eradicated from USA in 1889 (Soper 1960), in the United Kingdom 1898 (Ritchie 1959), and from Holland in 1887, Switzerland 1895, Hungary 1902 (Hutyra, Marek and Manninger 1946) and from South Africa in 1922 (Lambrechts 1962). The disease is enzootic in some parts of West Africa (FAO report 1967), (Blood and Henderson 1977), where its continual prevalence conforms with the definition of a true endemic or enzootic disease as "one in which social circumstances do not offer any effective barrier to its spread" (Burnet 1962). In Nigeria a conserva-
tive estimate of the cost of annual losses due to this single disease would amount to 3.6 million (± 2) naira. The purpose of this communication is to report the incidence of this disease in Nigeria and to examine the prospects of eliminating the disease from Nigeria.

PREVALENCE AND INCIDENCE OF CBPP IN NIGERIA
Griffin and Laing (1966) reported 26 outbreaks in 7 areas of Nigeria. The incidence of CBPP in Nigeria is not clear except for old Bornu and Kaduna Provinces where figures of 0.51% and 0.13% respectively have been reported (Ferguson 1964). Contagious bovine pleuropneumonia was reported rampant in the northern provinces of Nigeria during the early decades of the present century (Nigeria, 1927) and (Nigeria, 1931). In 1957, 229 outbreaks were recorded in Bornu province alone. Griffin and Laing (1966) plotted the epizootic curve of the outbreaks in Northern Nigeria during the period of 1953 - 1964 which showed the absence of CBPP in Nigeria by 1964. Karst (1970) regarded the disease as extinct in Nigeria by 1965. Since then there was recrudescence, reintroduction and reinfection during the last Nigerian Civil war when veterinary Services inevitably became slack. From the war years onwards to the postwar period (1966 - 1974), the result of laboratory examination of specimens sent to Diagnostic and Investigation Division of the National Veterinary Research Institute, Vom, showed a variable inci-
dence. The remarkable variation observed during this period was characterized by a rapid rise in incidence of 15.1% in 1966—70, from pre-war years when there was no CBPP in Nigeria to 30.2% in 1971. Since then the incidence had dropped to 4.6% by 1974. Table (1) gives details of the incidence and the number of confirmed outbreaks which occurred during this period under review.

Table 1: Trends in incidence of C.B.P.P. 1970—1974

<table>
<thead>
<tr>
<th>Year</th>
<th><strong>No. of specimens received</strong></th>
<th><strong>No. positive</strong></th>
<th><strong>% positive</strong></th>
<th><strong>No. of outbreaks</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>1970</td>
<td>2596</td>
<td>391</td>
<td>15.1</td>
<td>*</td>
</tr>
<tr>
<td>1971</td>
<td>4628</td>
<td>1399</td>
<td>30.2</td>
<td>63</td>
</tr>
<tr>
<td>1972</td>
<td>2810</td>
<td>594</td>
<td>21.1</td>
<td>33</td>
</tr>
<tr>
<td>1973</td>
<td>3442</td>
<td>312</td>
<td>9.1</td>
<td>19</td>
</tr>
<tr>
<td>1974</td>
<td>1998</td>
<td>94</td>
<td>4.6</td>
<td>*</td>
</tr>
</tbody>
</table>

* Figures of confirmed outbreaks are unavailable
** 3.2% of all specimens were found unsuitable for examination.

THE IMPROPER OF PRESENT CONTROL MEASURES.

In Nigeria, control measures against CBPP usually follows conventional procedures. Following confirmed outbreaks infected herds are slaughtered (FAO 1966) with ring vaccination and quarantine of adjacent herds being carried out. In some states there is compensation. Besides, in most states of the country there is annual vaccination using the T1 strain of M. mycoides. Indeed vaccination coverage judged by the number of the vaccine issued from the Veterinary Research Institute, Vom, Nigeria would appear to be very extensive. Although there is reduction in the incidence of CBPP, probably due to annual vaccination, the present control measures are without doubt inadequate because of the difficulty over the control of cattle movement within and into Nigeria (Davies 1966).

THE PROSPECTS AND PROBLEMS OF ERADICATION IN NIGERIA

In our view the eradication of CBPP in Nigeria could be achieved in two ways — namely by applying the test and slaughter policy and by mass vaccination. But considering the economy of our livestock industry, mass vaccination is preferable because immunization programmes have proved to be an efficient and relatively inexpensive method of disease control (Foeg and Eddins 1973) Nevertheless in most parts of the world where CBPP eradication had been achieved a long time ago, the eradication came through employing the slaughter policy (Lindley 1967).

The choice vaccine for this purpose is the T1 vaccine (Davies, Magiga, Shifrine and Read 1968). The administration of vaccines involves initial surveillance for bovine trypanosomiasis in order to avoid the immunosuppressive effect due to trypanosome infections (Godwin 1970). Such infections are encountered nowadays in areas thought free of tsetse fly (FAO 1966), (Osinyemi and Agbonlahor 1980). The National Veterinary Research Institute, Vom, issued out 16.5 million doses for the two years 1973 and 1974 for an estimated cattle population of 11 million (FAO 1966). The institute could meet field demand by a planned programme of production.
A national policy for the control of CBPP in Nigeria is essential. In this policy must be included international cooperation, because technical assistance may be sought from other countries in addition to soliciting the cooperation of neighbouring countries in this exercise.

CONCLUSION

The eradication herein advocated is the control of disease to the point of continued absence of transmission within a specified area (Andrews and Langmuir 1963). It is half a century ago that prophylactic vaccination was introduced in Nigeria (Griffin and Laing 1966). It is time the disease was eliminated so that no further effort of any kind is necessary to prevent return of infections.

The ultimate aim of preventive and control measures is to eradicate the disease (Gordon 1965). The advantages of eradication of CBPP are well known and when funds and facilities are provided the time would be deemed ripe for the final assault on the disease. It is hoped that eradication can be achieved within ten years of mass vaccination campaign programmes in Nigeria.

ACKNOWLEDGEMENTS

I wish to thank Messrs D.E. Agbonlahor, FIMLS, G.A. Adesimi, AIMLT and M. C. Ohaeri AIMLT for technical assistance and The Director National Veterinary Research Institute, Vom, for permission to publish.

REFERENCES


Received for publication on 16th August, 1977
SALMONELLA AND ESCHERICHIA COLI STRAINS ISOLATED FROM POULTRY IN IBADAN, NIGERIA.

S. FALADE and M.U.M. EHIZOKHALE
Faculty of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria.

SUMMARY

Four strains of Salmonella livingstone were isolated from three hundred samples of intestinal and gall bladder contents and faecal swabs from broilers at the University of Ibadan Teaching and Research Farm. Avian salmonellosis in this locality is briefly reviewed with comments on the serotypes involved. Two strains of E. coli, 0112: K66 and 0127: K63 were also isolated from one hundred samples examined.

INTRODUCTION

Poultry was incriminated as the commonest reservoir of salmonella in England by Savage (1956), while in the U.S.A., Edwards (1939) found that fowls were the greatest reservoir of paratyphoid infection. In a survey of the incidence of salmonella infections in Britain between 1968 and 1973, a total of 1548 cases were recorded of which 1225 were in poultry. (Sojka, Wray, Hudson and Benson, 1975). Brown, Duff, Wilson and Ross (1973) in a survey into the incidence of salmonellae in broilers and broiler breeders in Scotland isolated eleven serotypes of which S. typhimurium was predominant.

In Nigeria, scanty information is available on the serotypes of Salmonella affecting poultry. Sen and Collard (1957) found a carrier rate of 4.5% in the intestinal contents of two hundred chickens collected from kitchens of halls of residence of the University College, Ibadan. These were birds supplied by different food contractors from local markets in and around Ibadan and Ilorin provinces. Both workers isolated nine strains belonging to five serotypes namely: S. edinburgh (one strain), S. africana (one strain), S. hadar (four strains), S. rubislaw (one strain and S. breedeney (two strains). Salmonella hadar was found in 44% of the isolations. Collard and Sen (1960) also isolated S. mission from an ostrich egg. Olufemi, Etukudo and Falade (1979) isolated three strains at the University of Ibadan Teaching and Research Farm. Also of 49 salmonella strains (Groups A-G) isolated from clinical cases between 1971 and 1975 in this faculty, 15 were associated with enteric infections in poultry (Falade, Ojo and Ogunnariwo, 1977). The serotypes were not however determined. Recently, an attempt was made to screen the same farm for salmonellae and our findings are reported in this communication.

MATERIALS AND METHODS

A total of 300 broilers were included in the survey. Faecal swabs were taken from 250 live broilers. Intestinal and gall bladder contents were collected from 50 broilers slaughtered for consumption. All samples were cultured in selenite broth and incubated at 37°C for 24-48h; and sub-cultures made onto desoxycholate citrate agar (DCA). After further incubation for 24h, salmonella-like colonies were sub-cultured onto MacConkey agar medium. Non-lactose fermenting colonies were isolated and sub-cultured onto nutrient agar slants.
These isolates were tested by the slide agglutination method using salmonella polyvalent ‘O’ and ‘H’ antisera Wellcome Batch K1971 and K1630 respectively. Those positive were later sent to Dr. B. Rowe, Director Salmonella /Shigella Reference Laboratory, Colindale, U.K., for complete identification.

In addition, *Escherichia coli* colonies isolated from the intestinal contents of all slaughtered broilers and from randomly selected faecal swabs of fifty live broilers were tested for slide agglutination using Wellcome polyvalent 2,3, and 4 *E. coli* agglutinating sera. Positive strains were sent to Dr. J.R. Thomlinson of the Department of Veterinary Pathology, University of Liverpool, U.K. for complete serotyping.

**RESULTS**

Of the 300 samples examined, only 4 samples of intestinal contents yielded salmonella. The strains were identified as *S. livingstone* 6, 7: B: 1, W. Out of 100 *E. coli* strains tested, 96 strains were untypeable with all the polyvalent antisera. Four strains which reacted positively with polyvalent 4 antiserum only were identified as 0112: K66 (2 strains) and 0127: K63 (2 strains).

**DISCUSSION**

From our findings, the carrier incidence for salmonellae is very low (1.3%), and in general salmonellosis has not been a problem on this farm, as no outbreak has been reported over the past several years (Adesiyan, personal communication). On the other hand, it is common practice for the poultry assistants to treat sick birds showing signs of diarrhoea with antibiotics and coccidiostats, hence one might consider the antibiotic therapy as a limiting factor for the isolation of salmonella.

It is interesting to note that the common strains in poultry *S. gallinarum* and *S. pullorum* were not isolated, probably due to the fact that the survey did not include diseased birds. Similar observations were made by Plowright (1957) and Olufemi et al., (1979).

Recently, an outbreak of pullorum disease (Adene, personal communication) and another of fowl typhoid in 18-24 months old layers (Ojo, personal communication) were reported at Olaogun Farm — a private poultry establishment in Ibadan. *S. pullorum* and *S. gallinarum* were isolated respectively. Both the University and Olaogun Farms are intensively managed, but there could be more cases of latent and clinical salmonellosis (if investigated) amongst the private small-scale commercial and backyard poultry in Ibadan and the country as a whole. The number in this latter category has tremendously increased following the launching of OFN (Operation Feed the Nation) programme by the Federal Military Government in 1977. *Salmonella livingstone* which is reported here in poultry for the first time had been previously isolated from human faeces (five strains) and a healthy pig (one strain) by Collard and Sen (1960) and Sen and Collard (1957).

Prior to a previous report (Falade, 1977), there was no information on *Escherichia coli* strains associated with poultry infections in Nigeria. From the yolk sac of one hundred dead chick embryos on the University Farm, 18 *E. coli* strains belonging to serogroups 0141: K85a, c (B)—16 strains and 0139: K82 (B) — 2 strains were isolated. (Falade, 1977). From a further 227 yolk samples examined (Falade 1977,
unpublished data) serogroups 0112: K66 — 1 strain and 0114: K90 — 1 strain were isolated. From the present findings, it would appear that the E. coli typeable strains vary in both age groups. Further investigation is in progress.

ACKNOWLEDGEMENT

The authors are grateful to Dr. B. Rowe, Director, Division of Enteric Pathogens, Public Health Laboratory, Colindale, U.K. and to Dr. J.R. Thomlinson of the Department of Veterinary Pathology, University of Liverpool, U.K. for the assistance in typing the isolates.

REFERENCES


Received for publication on 24th April, 1978.
THE NORMAL SKIN BACTERIAL FLORA OF SOME CATTLE BREEDS IN NIGERIA

K.J. NWUFOH* and S.F. AMAKIRI,
Department of Veterinary Anatomy, University of Ibadan, Ibadan, Nigeria.

SUMMARY

Skin swabs from various body sites of clinically normal White Fulani, N'Dama and Friesian cattle were cultured and examined bacteriologically. Coagulase negative Staphylococcus saprophyticus (S. epidermidis), B — haemolytic Streptococcus, Escherichia coli and Bacillus species organisms were consistently seen.

Breed differences in the distribution of various bacterial types were noted. In the N'Dama, the Bacillus species were predominant with few staphylococcus organisms while in the Friesian and White Fulani, the latter was the most prevalent.

INRODUCTION

Considerable work has been done on the normal microflora of human skin and these have been adequately documented (Maibach and Hildick-Smith, 1965; Marples, 1965; Skinner and Carr, 1974; Noble and Somerville, 1974).

In animals, such studies have been confined mainly to the dog and cat (Kral and Schwartzman, 1964; Muller and Kirk, 1976; Krogh and Kristensen, 1976). In cattle, much of the earlier studies on skin micro-organisms have been concerned with pathogenic species until recently when Lloyd, Dick and Jenkinson (1979) reported on the normal skin microflora of bovines in temperate environments.

In tropical environments, many economically important skin diseases cause spoilage of hides. The need to investigate the pathogenic organisms which cause such infections necessarily brings into focus normal skin microflora which may affect the pathogenesis of the infection either as antagonists or synergists.

It is for this reason that the study of the normal skin bacteriology of some breeds of cattle in Nigeria was undertaken. The results obtained are reported in the present paper.

MATERIALS AND METHODS

Ten animals each of White Fulani, N'Dama and Friesian cattle were used in this study. Seven duplicate skin swabs (one from each side of the animals) were taken from the neck, back, rump, flank, ventral abdomen and the fore and hind limb regions of each animal in the dry season (November/December, 1978) and the rainy season (June/July, 1979). The animals were all housed in a pen and fed fresh grass and concentrates with water supplied ad libitum. Swab samples were placed in tryptose soya broth (TSB) for 24 h and later cultured on blood and nutrient agar under aerobic and anaerobic conditions, at 37 °C for 24 h, on MacConkey agar (aerobically), and for 48 h on blood agar under CO₂ enriched atmosphere for Dermatophilus congolensis (D. congolensis) isolation. Discrete colonies were later subcultured on blood agar twice to obtain pure culture of the organisms.
The morphology of the various organisms was later examined microscopically on staining by the Gram's technique.

Colonies which resemble staphylococcus species morphologically and microscopically were further tested for coagulase production with human plasma. Various biochemical tests were also carried out to confirm the identification of the organisms using the methods of Cowan (1974) and Buchanan and Gibbons (1974).

RESULTS

Bacterial organisms identified

The same type of bacterial organisms were seen on the various body sites examined in the different breeds of cattle, and no differences related to the season of sampling were observed.

The organisms identified in all cases in the various media used in this study included *Staphylococcus epidermidis* (S. epidermidis) β-haemolytic streptococcus, *Escherichia coli* (E. coli) and *Bacillus species* which all grew more under aerobic than in anaerobic conditions. No *D. congoensis* organisms were seen in the skin swabs taken from these healthy cattle.

Breed differences in organisms seen

The above listed organisms were seen in the normal skin of all the breeds of cattle sampled. The *Bacillus species* were the most predominant on the skin of the N'Dama cattle whereas *Staphylococcus* organisms were abundant in the Friesian and White Fulani skin swabs as judged by the number of colonies on the blood agar plates.

DISCUSSION

The organisms identified in this study were consistently present on all body sites of the breeds of cattle studied and also in the rainy and dry seasons. This indicates that they may be resident rather than transient inhabitants of the skin. The skin bacterial flora identified in cattle in this study is similar to that noted on the skin of man (Naylor, 1970; Noble and Sommerville, 1974) and in dogs and cats (Heast; 1967 a.b., Krogh and Kristensen, 1976) where the micrococci were the most common residents.

Lloyd et al. (1979) observed that the skin microflora of temperate cattle comprised mainly of bacteria, yeast and filamentous fungi. The different morphological groups of bacteria identified by them included cocci, pleomorphic rods an uniform rods (with morphology characteristic of the *Bacillus species*); their results are similar to our findings.

The breed differences in the distribution fo *Bacillus* organisms noted in this study are of particular interest especially as a *Bacillus species, B. subtilis* is known to produce subtilin — an antibacterial product and bacitracin, an antibiotic from the substance bacillin (Jensen and Hirschmann, 1944; Johnson, Anker and Meleney, 1945; Foster and Woodruff, 1946). The preponderance of these *Bacillus* organisms on the skin of the N'Dama, a breed of cattle resistant to many skin diseases including streptothricosis caused by *D. congoensis* (Coleman, 1967) may well be significant. Moreover, the fact that this organism is closely related to another organism — *Bacillus pumilus* (Buchanan and Gibbons, 1974), a contaminant recently identified as an inhibitor to *D. congoensis* and other micro-organisms in vitro (Ojo, 1975) strengthens the assumption that the high colonial population of *Bacillus species* on the N'Dama skin probably inhibits the growth of *D.
congolensis and other pathogenic organisms on the skin of this breed of cattle.

ACKNOWLEDGEMENTS

The authors are grateful to Professor M. Ola Ojo of the Department of Veterinary Microbiology and Parasitology, University of Ibadan, for his advice on methodology, critical examination of the slides and general interest in this study, and to Mr. M. A. Arasi for his technical assistance in the bacteriological work.

REFERENCES


Received for publication on 18th February, 1980
SEASONAL VARIATIONS IN COPPER LEVELS IN YANKASSA SHEEP IN ZARIA

E.O. GYANG, O. OGUNBIYI and M. HULL*
Faculty of Veterinary Medicine, A.B.U., Zaria, Nigeria.

SUMMARY

Copper levels in plasma, liver and hair were estimated in local Yankassa sheep (418 for plasma, 44 for liver and 27 for hair) grazing around Zaria in two dry seasons and two wet seasons. The respective Cu values for dry season and wet season were 1.56 ± 0.33 and 1.12 ± 0.23 for plasma, 13.65 ± 4.70 p.p.m. and 8.15 ± 3.14 p.p.m. for liver and 5.48 ± 1.34 and 4.48 ± 1.34 and 4.48 ± 1.74 p.p.m. for hair. The liver Cu levels were lower than those reported in sheep elsewhere. It is suggested that wet season Cu deficiency does occur in the area, and may be a factor in the widespread anaemia diarrhoea and unthriftiness observed in the area.

INTRODUCTION

Copper (Cu) was first shown to be essential for animal nutrition in 1928 (Lee, 1950). The signs of deficiency vary from dramatic muscular incoordination in lambs to mild non-specific symptoms such as emaciation, diarrhoea and anaemia in adults (Beck & Bennels, 1963). Other signs are depigmentation of hair and “stringy or steely wool” (Underwood, 1962). The role of the element in hematopoiesis and skeletal system has also been established (Jensen et al, 1958).

Often the clinical signs of the deficiency are non-specific and it may be difficult to recognise Cu deficiency.

This is especially so in wool sheep, in which the “stringy wool” cannot be seen.

In Northern Nigeria non-specific signs like anaemia, depressed appetite and emaciation are observed. This is evidence to suggest that factors, apart from parasitism, are responsible for anaemia and emaciation in sheep grazing in Zaria area. The purpose of this investigation is to determine the Cu status of the Yankassa sheep in Zaria.

MATERIALS AND METHODS

Plasma

Plasma samples were obtained from 418 adult animals grazing on local unimproved pastures within a radius of 26 miles around Zaria from October, 1973 to September, 1975. All samples were obtained by jugular puncture into plain vacuum venoject tubes. In the laboratory the samples were centrifuged within 12 hours of collection. The plasma obtained was stored at 4°C and plasma Cu levels were determined using Atomic Absorption Spectrophotometre Model 209B at 3247A, using the Perkins and Elmer method (Perkin-Elmer, 1968).

Liver Copper

Whole liver samples were obtained from 44 normal adult sheep; slaughtered for this study; 21 in the dry season and 23 in the wet season. The gall bladder was dissected out immediately and the liver kept frozen till analysed. One gram of each liver sample was wet-
ashed and levels of Cu determined (Perkin-Almer, 1968).

Hair Copper

Hair samples were obtained by shaving at the withers of 27 sheep grazing in Zaria area; 13 in the dry season and 14 in the wet season, and analyzed for Cu (Matsuba et al., 1970).

RESULTS

The results of the Cu values are shown in Tables 1 & 2. The dry season plasma Cu values ranged from 0.80 to 3.80 p.p.m., with a mean value of $1.56 \pm 0.33$ p.p.m., while the wet season value ranged from 0.74 to 1.72 p.p.m with a mean of $1.12 \pm 0.23$ p.p.m. The dry season liver Cu values ranged from 5.50 to 29.50 p.p.m. with a mean of $13.65 \pm 4.70$ p.p.m. The wet season liver Cu values with a range of 3.50 to 18.00 p.p.m., and a mean of $8.15 \pm 3.14$ p.p.m., were significantly $P < 0.1$ lower than those of the dry season. The mean for the hair copper levels were $5.48 \pm 1.34$ p.p.m. and $4.48 \pm 1.74$ p.p.m for dry season and wet season respectively.

Table 1: Monthly variation in serum Cu levels

<table>
<thead>
<tr>
<th>Month</th>
<th>No. of animals</th>
<th>Range p.p.m.</th>
<th>Mean &amp; S.D. (p.p.m.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>25</td>
<td>0.80 – 1.50</td>
<td>1.29 ± 0.17</td>
</tr>
<tr>
<td>February</td>
<td>27</td>
<td>1.20 – 3.80</td>
<td>1.99 ± 0.17</td>
</tr>
<tr>
<td>March</td>
<td>42</td>
<td>1.10 – 2.20</td>
<td>1.67 ± 0.46</td>
</tr>
<tr>
<td>April</td>
<td>33</td>
<td>1.30 – 2.60</td>
<td>1.82 ± 0.41</td>
</tr>
<tr>
<td>May</td>
<td>50</td>
<td>0.80 – 1.72</td>
<td>1.02 ± 0.29</td>
</tr>
<tr>
<td>June</td>
<td>29</td>
<td>0.80 – 1.62</td>
<td>1.21 ± 0.24</td>
</tr>
<tr>
<td>July</td>
<td>35</td>
<td>0.96 – 1.24</td>
<td>1.09 ± 0.9</td>
</tr>
<tr>
<td>August</td>
<td>37</td>
<td>0.74 – 1.63</td>
<td>1.15 ± 0.27</td>
</tr>
<tr>
<td>September</td>
<td>35</td>
<td>0.80 – 1.72</td>
<td>1.29 ± 0.26</td>
</tr>
<tr>
<td>October</td>
<td>21</td>
<td>1.10 – 2.10</td>
<td>1.52 ± 0.23</td>
</tr>
<tr>
<td>November</td>
<td>18</td>
<td>1.18 – 1.60</td>
<td>1.31 ± 0.14</td>
</tr>
<tr>
<td>December</td>
<td>27</td>
<td>0.96 – 1.64</td>
<td>1.30 ± 0.22</td>
</tr>
</tbody>
</table>
Table 2: Copper values in plasma, liver and hair for dry and wet seasons

<table>
<thead>
<tr>
<th>Tissues</th>
<th>Dry Season (October — March)</th>
<th>Wet season (April — September)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Animals</td>
<td>Mean &amp; SD.</td>
</tr>
<tr>
<td>Plasma</td>
<td>212</td>
<td>1.56 ± 0.33 p.p.m</td>
</tr>
<tr>
<td>Liver**</td>
<td>21</td>
<td>13.6 ± 4.70 p.p.m</td>
</tr>
<tr>
<td>Hair</td>
<td>13</td>
<td>5.48 ± 1.34 p.p.m</td>
</tr>
</tbody>
</table>

**Seasonal Means significantly (P < 0.01) different.

DISCUSSION

Normal plasma Cu levels in sheep is reported to be between 0.80 to 2.00 p.p.m. (Albiston, Bull, Dick and Keast, 1940; McCostner, 1968). This would suggest that both the dry season and the wet season values of plasma Cu in Yankassa sheep grazing around Zaria are within the normal range reported for the specie. The wet season values are however lower than those obtained for the dry season. Normal liver copper in sheep has been reported to be about 12.20 p.p.m. (Eden, 1940). This would suggest that the liver copper levels of Yankassa sheep grazing around in the dry season are within the normal range (13.65 ± 1.34 p.p.m.), but the wet season liver Cu values (8.15 ± 3.14 p.p.m.) are lower than those reported for sheep. No work appears to have been done on hair Cu values in sheep hair. In this survey we found the mean hair copper values for sheep to be 5.48 ± 1.34 p.p.m.

The low plasma and liver Cu values during the wet season would suggest that a marginal Cu deficiency does exist in sheep grazing during the rainy season around Zaria and may be a contributory facotr in the anaemia and unthriftiness seen in sheep in Zaria area during the rainy season.

REFERENCES


Received for publication on 23rd May, 1979
MALIGNANT CATARRHAL FEVER VIRUS SHEDDING BY INFECTED CATTLE

E.Z. MUSHI and F.R. RURANGIRWA
Veterinary Research Department, Muguga, Kenya Agricultural Research Institute, P.O. Box 32, Kikuyu, Kenya.

An important feature of malignant catarrhal fever virus (MCFV) infection of cattle is that the infection is not contagious, for sick and normal cattle can be housed or grazed together with impunity (Mettam 1923; Piercy 1952; Plowright 1964; Kalunda 1975). Rabbit-to-rabbit transmission by contact has also not been reported in spite of the fact that these animals have been extensively used for experimental infections (Plowright, 1964). In this context cattle and rabbits can be considered “dead-end” hosts to MCFV. However, in wildebeest calves, MCFV is shed in a cell-free state in nasal and ocular secretions and this virus is transmitted amongst wildebeest and occasionally into cattle via these secretions (Rweyemamu, Karstad, Mushir, Otema, Jessett, Rowe, Drevemo and Grootenhuis, 1974; Mushir, Karstad and Jessett, 1980). Although MCFV has been isolated from nasal and oral secretions of MCFV infected cattle (Kalunda, 1975) the state in which the virus was shed, that is whether cell-free or cell-associated was not established. The purpose of this investigation therefore was to test MCFV infected cattle secretions for cell-free virus.

Grade steers which were 3 years old were inoculated intravenously with bovine thyroid (BTh) grown cell-associated MCF virus which had been isolated from wildebeest nasal secretions. Nasal, ocular and oral secretions were collected on cotton wool swabs and transported in phosphate buffered saline solution containing 0.1% bovine albumin and antibiotics. The unclarified secretions were inoculated onto confluent BTh cell cultures and observed for the development of cytopathic changes (cpe) for 21 days. Supernatants from secretions which had been centrifuged at 400 g for 10 minutes were also assayed for virus infectivity.

MCFV was isolated from the unclarified ocular and nasal secretions collected from 5 out of 5 MCF sick steers. The mean detected infectivity titres of the nasal and ocular secretions were $10^{1.13}$ and $10^{1.65}$ TCID$_{50}$/ml respectively (Table 1). No virus was isolated from oral swabs. The infectivity in the ocular secretions persisted throughout the course of disease but once the nasal secretions became mucopurulent the virus could no longer be isolated. The main cell type in these secretions was epithelioid. MCFV was not detected in the supernatant fluids of centrifuged cattle secretions (Table 1) thus one cycle of light centrifugation was able to deposit all the infectivity.

MCFV was demonstrated in the cellular components of nasal and ocular secretions of the infected cattle unlike in wildebeest secretions where the virus occurs in a cell-free and cell-associated state. The failure to isolate this virus from oral secretions in the current study could be due to the copious saliva which dilutes the infectivity. MCFV was not isolated from nasal secretions which were mucopurulent. Kalunda (1975) attributed this to the
fact that such secretions were toxic to the cell cultures. In the present study these secretions were not toxic to the cell cultures, for inoculated monolayers remained healthy for 21 days, most likely, the infectivity was destroyed by immune mechanisms. Preliminary work has demonstrated virus neutralising antibodies in nasal secretions of an MCF infected steer. The antibodies were detected during the terminal stages of the disease (Mushi, unpublished).

Table 1: MCF virus shedding by infected cattle.

<table>
<thead>
<tr>
<th>Animal No.</th>
<th>Day of Disease</th>
<th>Unclarified secretions</th>
<th>Supernatant</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>NS</td>
<td>OS</td>
</tr>
<tr>
<td>N153</td>
<td>3</td>
<td>NT</td>
<td>1.2</td>
</tr>
<tr>
<td>N370</td>
<td>3</td>
<td>1.0</td>
<td>0.5</td>
</tr>
<tr>
<td>N148</td>
<td>3</td>
<td>0.9</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>Nil</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>&quot;</td>
<td>2.2</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>&quot;</td>
<td>2.5</td>
</tr>
<tr>
<td>N424</td>
<td>8</td>
<td>&quot;</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>&quot;</td>
<td>1.8</td>
</tr>
<tr>
<td>N453</td>
<td>5</td>
<td>1.5</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Nil</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>Nil</td>
<td>1.5</td>
</tr>
</tbody>
</table>

NT: Not Tested  
NS: Nasal Secretion  
OS: Ocular Secretion  
SAL: Saliva

In contrast, virus neutralising activity was not detected in the ocular and oral secretions. Probably the virus neutralising antibodies, with the help of other local immune mechanisms were responsible for the removal of the infected cells from the nasal secretions in the terminal stages of the disease.

All the infectivity in cattle secretions was deposited by light centrifugation, suggesting that the virus was cell-associated and there was no stable cell-free virus in the secretions. The inability of MCFV to spread by contact in cattle is probably due to the absence of cell-free virus in these secretions.

ACKNOWLEDGEMENT

This paper has been published with the permission of the Director, Veterinary Research Department, Muguga, Kenya Agricultural Research Institute.

REFERENCES


Received for publication on 10th December 1979
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30. Effect of solubel factors from Mycoplasma mycoides subsp. Mycoides on the collagen content of bovine connective tissue.
31. The toxicity of Capparis tomentosa in goats.

AUTHORS’ SUMMARY: The present study briefly reviews our preliminary breeding programme for resistance to leptospirosis in pigs. The percentage of progeny with positive results from matings between resistant boars and sows was very small (2.2%). The percentage of progeny with positive results from mating between susceptible boars and sows was very high (67.1%). In resistant pigs the level of leptospira antibodies was low (from 1: 100 to 1: 200) and it was very high in susceptible pigs (up to 1: 12800). The $h^+$ of leptospirosis resistance was 0.20 to 0.21. Association of leptospirosis resistance with economic traits in sows was also analysed.


AUTHOR’S SUMMARY: A sporadic natural infection of sheep with *Brucella abortus* in a sheep breeding centre in Kano State, Nigeria, is reported. Sera and milk samples from the flock of Sudanese long- and fat-tailed sheep used for cross-breeding purposes were examined for brucellosis by the Rose Bengal Plate Test (RBPT), serum agglutination test (SAT) and Milk Ring Test (MRT) respectively. The overall positive retractor rate to SAT was 14.5%. All 22 ewe milk samples examined by MRT were positive and *Brucella abortus* was cultured from 5 of the 22 ewes samples. This is believed to be the first report of bacteriological isolation of *Brucella* in sheep in Kano, Nigeria.


AUTHORS’ SUMMARY: Three groups of five piglets were formed and 1390 *Escherichia coli* isolates were obtained during the 45-day period of observation. One of the groups received feed without antibiotic whereas the second received feed containing 100 ppm neomycin and the third feed with 100 ppm neomycin plus 100 ppm tetracycline. Rectal swabblings for bacterial isolation were repeated ten times, twice during an adaptation period and eight times during the treatment period. Resistance among the isolates to tetracycline, streptomycin and triple sulfas remained high throughout this experiment whereas resistance to neomycin, chloramphenicol and ampicillin were found to increase significantly under the influence of antibiotic supplemented feed. This increase of antibiotic resistance was associated with an increase of the percentage of isolates harbouring an R. factor. When comparing the ability of strains harboring an R. factor to receive the plasmid Ent from the *E. coli* K12 (P15) with isolates not harbouring such a plasmid, no significant difference was observed in their ability to receive the Ent plasmid.


AUTHORS’ SUMMARY: When inoculated by various routes with approximately $10^8$ (or $10^7$) *Brucella canis* organisms, all adult dogs became infected. When dogs were inoculated with approximately $10^6$ organisms by different routes, the positive rate of infection by the route of inoculation was as follows: 2/2 (No. infected/No. inoculated) (intravenous), 2/2 (subcutaneous), 2/4 (conjunctival), 0/4 (oral) and 0/4 (intravaginal). Five male dogs were inoculated orally with 5.2 x $10^8$ *B. canis* organisms and examined weekly for urine. The organisms were first detected in their urine a few weeks after the onset of bacteremia. Then a high concentration of these organisms was usually excreted continuously. Five 4-week-old puppies born from a *B. canis*-free bitch were housed together with these infected males in an isolated facility for 18 weeks. One of them became infected. The transmission of brucellosis to the puppy seemed to have been caused by a contact with *B. canis*-contaminated urine.

IBAR/1981 de I.N. KANTOR, MAR-5 CHEVSKY, N. and LOMBARDI, R.
Tuberculin Response Related to the Administration of Foot and Mouth Disease, Brucellosis and Rabies Vaccines.


AUTHORS' SUMMARY: Experiments have been carried out to determine whether vaccinations against foot and mouth disease, brucellosis and rabies have any influence on the levels of response to the tuberculin test when carried out simultaneously or shortly before this test. In experiments in guinea pigs, a significant decrease in tuberculin reactions was observed after foot and mouth disease vaccination. However, in experiments in cattle, vaccination did not appear to interfere with the tuberculin test.

IBAR/1981 CONDY, J.B.

6 A History of Foot and Mouth Disease in Rhodesia.

*Rhodesian Vet. J.*, 1979, 10: 2–10

AUTHOR'S SUMMARY: The history of foot and mouth disease in Rhodesia is described from its first appearance in that country in 1892 to the outbreaks of 1978. Details of the 62 primary foci of disease which occurred between 1931 and 1978 are given in tabular form. The evolution of disease control policies since 1931 is considered, as is the history of wildlife involvement in the disease situation.

IBAR/1981 FAGBAMI, A.H.

7 Susceptibility of West African Dwarf Sheep to the Indigenous Wesselsbron Virus.


AUTHOR'S SUMMARY: West African Dwarf sheep infected with Wesselsbron virus developed clinical disease characterized by fever anorexia, mild leucopenia and abortion. All inoculated animals developed viraemia lasting three to four days. Haemaggulutination inhibiting and neutralizing antibodies to Wesselsbron and related flaviviruses were detected in all infected animals.

IBAR/1981 LEFEVRE, P.C., BONNET, J.B. and VALLAT, B.

Lumpy Skin Disease. I. Epizootioloigies in Africa.


AUTHORS' SUMMARY: The authors draw the reparation map of lumpy skin disease in Africa with emphasis on the dynamic of extension of the disease. The case of Chad is examined; after the 1973 epizootic, the disease is now enzootic.

IBAR/1981 BRUNDAGE, L.J., DERBYSHIRE, J. B. and WILKIE, B.N.

Cell Mediated Responses in a Porcine Enterovirus Infection in Piglets.


AUTHORS' SUMMARY: Sixteen pathogen-free piglets were infected orally with porcine enterovirus strain T80 and the cell mediated response to the virus was measured at intervals after infection. Five uninfected piglets were used as controls. Indirect macrophage migration inhibition tests were performed with lymphocytes from blood, ileal lamina propria and mesenteric lymph node. Blood lymphocyte culture supernatants showed no consistent T80 specific effect on macrophage migration, suggesting the absence of a systemic cell-mediated response. Ileal lamina propria lymphocyte culture supernatants showed irregular migration stimulation. The mesenteric lymph node lymphocyte culture supernatants produced migration inhibition at seven days postinfection, followed by stimulation of migration from 10 to 22 days postinfection. Subsequently, migration was again inhibited from 24 to 31 days postinfection. It was concluded that the cell mediated responses in this infection were weak and localized and not associated with significant antiviral activity.

IBAR/1980 MOLOEN, R.H., ROWLANDS, D.J. and BROWN, F.

Comparison of the Antibodies Elicited by the Individual Structural Polypeptides of Foot and Mouth Disease and Polio Viruses.

*J. Gen. Virol., 1979, 45: 761–763*
AUTHORS' SUMMARY: Antibody produced in guinea pigs against preparations of the VP1 structural polypeptide of foot and mouth disease virus neutralised the virus and reacted with both full and empty particles in radioimmunoassays. Antiserum to VP2 reacted with artificial empty particles of the virus but not with full particles. On the other hand, none of the individual polypeptides of poliovirus produced antisceras which neutralised the virus nor reacted with it in radioimmunoassays. When poliovirus was disrupted into empty capsids, both VP1 and VP2 reacted in radioimmunoassays, indicating a conformational change in the capsid particles.

IBAR/1981 MAYR, A.
11 New Emerging Viral Zoonoses

*Veterinary Record, 1980, 106 (24): 503 – 506*

AUTHOR'S SUMMARY: New developments in the field of viral transmission from animal to man can be divided into four areas of study. First are the new viral zoonoses such as diseases caused by rotaviruses, Lassa virus and the animal orthopox viruses which will be more prevalent after the cessation of mandatory vaccination against smallpox. Secondly are the numerous ubiquitous viruses, such as adeno and herpesviruses, which in healthy animals lead only to clinically inapparent infections. A typical example of the third area is the recombination and hybridisation between animal and human influenza type A viruses. The final area is concerned with the transmission of viral zoonoses to man through food of animal origin.

IBAR/1980 HARRIS, T.J.R.
12 The Nucleotide Sequence at the 5' End of Food and Mouth Disease Virus RNA.

*Nucleic Acids Res., 1979, 7 (7): 1765 – 1786*

AUTHOR'S SUMMARY: Foot and mouth disease virus RNA has been treated with RNase H (from *E. coli*) in the presence of oligo (dg) specifically to digest the poly (C) tract which lies near the 5' end of the molecule. The short fragment containing the 5' end of the RNA was separated from the remainder of the RNA by gel electrophoresis. RNA ligase mediated labelling of the 3' end of the short fragment showed that RNase H digestion gave rise to molecules that differed only in the number of cytidylic acid residues remaining at their 3' ends and did not leave the unique 3' end necessary for fast sequence analysis. As the 5' end of the short fragment is blocked by VP, the S fragment was prepared from virus specific messenger RNA which does not contain this protein. This RNA was labelled at the 5' end using polynucleotide kinase and the sequence of 70 nucleotides at the 5' end determined by partial enzyme digestion sequencing on polyacrylamide gels. Some of this sequence was confirmed from an analysis of the oligonucleotides derived by RNase T1 digestion of the short fragment. The sequence obtained indicated that there is a stable hairpin loop at the 5' terminus of the RNA before an initiation codon 35 nucleotides from the 5' end. The RNase T1 analysis suggests that an 11 nucleotide inverted complementary repeat of a sequence near the 3' end of the RNA is present at the junction of the S fragment and the poly(C) tract.

IBAR/1981 DARGIE, J.D.


AUTHOR'S SUMMARY: Results are presented of a comparative study of the pathogenesis in the anaemia, hypo-albuminaemia and body weight changes occurring in N'dama and Zebu cattle infected with *Trypanosoma congolense*. The kinetics of the anaemia were examined through measurements of plasma and red cell volumes and rates of haemoglobin breakdown and synthesis, and measurements of albumin pools and catabolic rate provided an insight into the cause of the hypoalbuminaemia. The possible impact of reduced feed intake on both features, as well as on the pathogenesis of the accompanying changes in bodyweight, was assessed indirectly through measurements of water turnover.

IBAR/1981 ACHUTHAN, H.N., MAHADEVAN, S. and LALITHA, C.M.
14 Studies on Developmental Forms of Babesia bigemina and Babesia canis in Ixodid Ticks

AUTHORS’ SUMMARY: Examination of naturally infected adults of Boophilus microplus and Haemaphysalis bispinosa revealed bodies indistinguishable from sporozoites of Babesia bigemina. Examination of artificially infected larva, nymph and adult of Boophilus microplus and larva and nymph of B. annulatus showed bodies referable to the sporozoites of B. bigemina. Larva, nymph and adult of Rhizophalus sanguineus showed forms similar to the developing forms of Babesia canis. Nymph of Rhizophalus turanicus also revealed similar bodies.

Studies on the Anaemia in Rabbits Infected with Trypanosoma brucei brucei


AUTHORS' SUMMARY: Splenectomy delayed the onset of the anaemia in rabbits infected with either Trypanosoma brucei brucei S42 or T. b. brucei 427 and, particularly in the latter infection, also lessened its severity.

Parasitaemias were higher in the 427 infection although, terminally, massive parasitaemias were recorded in the S42 infection.

With \textsuperscript{51}Cr-labelled red cells, it was shown that there was an increase in both splenic sequestration and destruction of red cells in infected animals relative to controls, the degree of sequestration probably being related to the degree of splenic enlargement.

Red cell T\textsubscript{S0} values were virtually unaffected by trypanosomal infection of splenectomised rabbits, and in those splenectomised in mid-infection a marked increase in T\textsubscript{S0} was observed shortly after the operation.

Since the anaemia which eventually developed in the splenectomised animals was not accompanied by a reduction in T\textsubscript{S0}, it is unlikely to be of haemolytic origin. Other possible causes are considered.

IBAR/1980 LEWIS, D., PURNELL, R.E and BROCKLESBY, D.W.
Babesia divergens: Protection of Intact Calves Against Heterologous Challenge by the Injection of Irradiated Pirolasms.

*Veterinary Parasitology*, 1980, 6 (4): 297 – 303

AUTHORS’ SUMMARY: Blood from a splenectomized claf infected with Babesia divergens was divided into 20 ml aliquots which were \textit{r} irradiated at doses of 0, 24, 28, 32, 36 and 40 krad and then inoculated into groups of three intact calves. Animals receiving non-irradiated blood had typical mild B. divergens reactions, but those receiving irradiated blood had either very mild reactions or no overt reaction. When the calves were challenged with a similar number (7.5 \times 10\textsuperscript{7}) of heterologous parasites of a recently-isolated field strain, those which had received blood irradiated at 0, 24, 28 and 32 krad were all immune whereas four of the five surviving animals which had received blood irradiated at 36 or 40 krad were susceptible. The immune status of individual cattle was reflected accurately in the results of the micro-ELISA test, which detected a significant rise in serum antibody titre of the four susceptible animals by day 21 after challenge.

IBAR/1980 SANDEMAN, R.M. and HOWELL, M.J.

\textit{In vitro} Studies of the Response of Sheep to Infection with Fasciola hepatica.

*Veterinary Parasitology*, 1980, 6 (4): 347 – 357

AUTHORS’ SUMMARY: When excysted metacercariae of Fasciola hepatica were cultured in serum from infected sheep a precipitate formed on their teguments and in the medium. This precipitate was found to contain sheep antibody and parasite antigen. Immunodiffusion tests suggested that only one antigen antibody interaction was involved in the formation of precipitate.

The extent of precipitate formation \textit{in vitro} was examined in serum samples taken weekly from sheep for 20 weeks following infection with \textit{F. hepatica}. Comparisons of the amount of precipitate formed with the levels of liver (GLDH) and bile duct (\textit{Y}-GT) enzymes in the serum indicated that the antibody response reached its peak when juvenile flukes were causing most damage to the liver parenchyma as a result of their migratory activity. Antibody levels fell as the flukes became established in the bile duct.

The Immunosuppressive Effects of Experimental T. congolense Infections in Goats.

AUTHORS' SUMMARY: The agglutinin response of four groups of goats inoculated with *Brucella melitensis* vaccine, 0, 1, 2 and 4 weeks following experimental infection with *Trypanosoma congoense* was compared with that in non-infected controls. Four weeks after vaccination the goats were treated with a trypanocidal drug and the recovery of the immune response observed. The results indicated that the majority of animals had a significantly but not completely suppressed antibody response. This was most marked in the group vaccinated 2 weeks post-infection, which corresponded with the onset of parasitaemia. Although the mortality rate in the infected goats was high the titre in those remaining animals that were treated with the trypanocidal drug increased immediately after treatment. The possible implications of trypanosome induced immunosuppression for vaccination programmes in goats are discussed briefly.

IBAR/1981 KIMETO, B.A.
19 Fine Structure of *Theileria parva* in the Bovine Skin.

AUTHOR'S SUMMARY: Ultrastructural studies of *Theileria parva* in the bovine skin revealed 'infective particles' of the parasite. These parasite forms were pleomorphic and were found extracellular or within host lymphoid cells, neutrophils and erythrocytes. The parasites were a product of extracellular schizogony. They were phagocytosed by the host leukocytes but seemed actively to invade the erythrocytes. Several extracellular uninucleate schizonts were also observed. The presence of extracellular infective particles, uninucleate schizonts and multiciliate schizonts, some showing schizogony, suggests an extra-cellular life cycle of *T. parva* within bovine tissue.

IBAR/1981 SCHILLHORN VAN VEEN
T.W., FOLARAMNI, FOLA-
D.O.B., USMAN, S. and
ISHAYA, T.
Incidence of Liver Fluke Infections (*Fasciola giganticata* and *Dicrocoelium hospes*) in Ruminants in Northern Nigeria.

AUTHORS' SUMMARY: One thousand and twenty-four cattle, 550 sheep and 1,748 goats slaughtered in a rural slaughter slab during 1973 to 1975 were examined for evidence of liverfluke infections. The prevalence rate of *Fasciola giganticata* and *Dicrocoelium hospes* infections was respectively 65.4% and 56.0% in cattle, 40.8 and 15.1% in sheep and 17.6 and 5.2% in goats. Other trematodes detected were *Schistosoma bovis* and paramphistomes. The seasonal incidence of *F. giganticata* as well as of *D. hospes* was highest during and directly after the rainy season. The lower prevalence rate of *F. giganticata*, especially in the younger animals, during 1973–1974 was thought to be related to the 1973 drought. This was supported by the low prevalence rate in the long-range trade cattle which originated from drier areas. The results are discussed in relation to the climatic conditions during the survey period, as well as to the difference in epidemiology of *F. giganticata* and *D. hospes* infections in northern Nigeria.

IBAR/1981 COHEN, R.D. H.
21 Phosphorus in Rangeland Ruminant Nutrition: A Review.

AUTHOR'S SUMMARY: The role of phosphorus in ruminant nutrition is discussed with reference to factors influencing phosphorus turnover. Recent information leading to a re-estimation of P requirements is presented. Particular reference is made to recent information on the hormonal control of P homoestasis and its relationship to the Ca – P interaction. Emphasis is placed on the interaction of P with other nutrients, particularly Ca, protein and energy, and it is stressed that phosphorus cannot be considered as an isolated nutrient and panacea for ill-thrift and infertility of rangeland cattle and sheep.

IBAR/1981 WILLIAMSON, P., HEN-
NESSY, D.P. and CUTLER, R.
The Use of Progesterone and Oestrogen Concentrations in the Diagnosis of Pregnancy, and in the Study of Seasonal Infertility in Sows.

AUTHORS' SUMMARY: Plasma concentrations of progesterone and oestrogen in sows 18 days after mating were examined, firstly, as a method of pre-
gnancy diagnosis, and secondly, for indication of any abnormal endocrine patterns that may have been involved in the etiology of seasonal infertility in sows. Progesterone levels were the most relevant indicator of ovarian function. For the diagnosis of pregnancy, progesterone levels were accurate (97%), but for the diagnosis of non-pregnancy, especially where a high degree of early embryonic death and/or cystic follicles was suspected, as in this trial, progesterone levels were not as accurate (60%). Oestrogen levels were of use only in confirming the type of infertility suffered by non-pregnant sows. This study shows that seasonal infertility in mated sows in a complex of several forms of infertility, each manifest in a common physical symptom of delayed return to oestrus after a normal mating to a fertile boar. The forms of infertility involved appear to be early embryonic death, luteinized ovarian cysts, small ovarian cysts and silent oestrus.


AUTHOR’S SUMMARY: The time at which oestrus first occurred in sows after weaning was studied. The frequency of “silent heat” in primiparous and pluriparous sows was investigated by post-mortem examinations. The influence of various factors on the length of time elapsing between weaning and first oestrus was assessed on the basis of environmental data and data from individual sow records.


AUTHOR’S SUMMARY: Using a well-known equation relating the heat production to the respiratory measurements, the total heat produced in the animal is partitioned into heat produced in the rumen and heat produced in the body. It is shown that the values of the partition constants can be calculated by making two simple logical deductions. Consideration of known heats of fermentation of carbohydrate and protein shows that the deductions are justified.


AUTHOR’S SUMMARY: The influence of nutritional factors on the productivity of traditionally managed White Fulani cattle herds on the Jos plateau is described as part of a 2½ year study. The observations made point to the effects of the poor nutrition obtained from natural fodders during the dry season as well as to gross overgrazing. The effect of limited dry season supplementation on productivity is illustrated.


AUTHORS’ SUMMARY: An experiment was conducted to compare carcass composition and economic gains made from finishing range-fed Western Sudan Baggara bulls on a traditional and a proposed feeding method. The traditional method resulted in a non-significantly (P > 0.05) greater mean daily gain over a feeding period of 90 days and slightly heavier final feedlot weight than the proposed alternative. The traditional finishing method resulted in fatter carcasses with less muscle tissue than the proposed finishing method. Financial considerations indicated that finishing these bulls on the proposed feeding method would result in a greater profit to the cattle feeder than on the traditional method.


AUTHOR’S SUMMARY: A simple technique for the collection of brain samples for the post-mortem diagnosis of heartwater (*Gowdria ruminantium* infec-
tion) is described. A sharp spoon (curette) or teaspoon and a knife are the only instruments required. After the head has been removed from the body a sample of cerebellar cortex is collected with the spoon through the foramen occipitale, thus obviating the need for opening the skull itself. This diagnostic technique appears to be as reliable as the conventional technique of collecting samples from the cerebral cortex.

IBAR/1981 CAZIEUX, A., DUCRET, J. and MIRANDE-DUCRET D.
Practical Use of Cryosurgery in the Animal.


AUTHORS’ SUMMARY: Tissue freezing due to liquid nitrogen application can be used to treat, without general nor local anesthesia, superficial tumors (papillomas, perianal adenomas . . . ) ill-hearing wounds (exuberant granulations, fibrosis . . .) anal fistulas and many other diseases. Healing is always satisfactory and brings a good restoration of tissue local qualities. If cryonecrosis extends too deeply, pigmentation and pilosity won’t return.

Freezing units are now simple materials, and cryosurgery takes place advantageously among the different methods of exeresis in the animal.

IBAR/1981 OJO, M., KASALI, O.B. and OZOYA, S.E.


AUTHORS’ SUMMARY: The caprine strain (lb9) of *M. mycoides*, given by endobronchial inoculation, is capable in inducing infection in calves. Typical CBPP lesions were produced in imported breeds when they were simultaneously infected with a mouse passed strain of *T. vivax*. *Mycoplasma* persisted in the regional lymph nodes of 4 of the 5 infected animals which failed to develop lung lesions. In these animals the caprine strain might behave like a vaccine strain. Further studies are required as to the pathogenicity and immunogenicity of the caprine strain of *M. mycoides* for cattle.

IBAR/1981 BUTTERY, S.H., COTTEW, G.S. and LLOYD, L.C.

30 Effect of Soluble Factors from *Mycoplasma mycoides* Subsp. *Mycoplasma* on the Collagen Content of Bovine Connective Tissue.


AUTHORS’ SUMMARY: The amount of connective tissue produced by diffusion chambers containing liquid culture of *Mycoplasma mycoides* subsp. *mycoides* implanted intramuscularly in immune cattle was determined by the concentration of hydroxyproline in the tissue adjacent to the filter membranes. More was produced by these chambers than those containing medium alone or those with cultures of each of 3 other mycoplasma strains from cattle. There was some indication that the galactan produced by *M. mycoides* might be responsible. The connective tissue capsule was similar to that seen around sequestered necrotic tissue in natural cases of contagious bovine pleuropneumonia.

IBAR/1981 AHMED, O.M.M. and ADAM, S.E.I.

31 The Toxicity of *Capparis tomentosa* in Goats.


AUTHORS’ SUMMARY: Eleven Nubian goats were given daily oral doses ranging from 0.05 to 5 g per kg day of the dried leaves of *Capparis tomentosa* and died or were killed in extremis at various times after dosing. The main signs of poisoning were inappetence, muscular weakness, incoordination of movement, pain in the sacral region, dragging of the hind limbs, pallor of the visible mucous membranes and recumbency. An increase in the activity of GOT, in the concentrations of ammonia, sodium and potassium and a decrease in total protein and chloride were detected in the serum. Haematological changes indicated the development of hypochromic erythrocytopaenia. The outstanding pathological changes were peri-neuronal spaces in the grey matter of the spinal cord, cnetrilobular hepatocellular necrosis and degeneration or cecrosis of the cells of the renal convoluted tubules.
STAFF LIST

Director

P.G. Atang, BVMS (Glas.) DTVM (Edin.) MRCVS

Chief Animal Health Officer

A. G. Tall, Docteur Vétérinaire (Maisons-Alfort)

Scientific Officer


Livestock Officer

K.O. Adeniji, B.Sc. Hons. (Ife)

Documents Officer

Miss M.A.S. Ougo, B.A. Hons (Nairobi), Dip. Interprétariat et Traduction (Paris)

Translator

Mr. P. Somda